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NOVEMBER

# THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION

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Hornification of the Gums—*Orban*
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Inlay Casting—*Taylor and Paffenbarger*
Dental Economics—*Rhobotham, Dittmar and Logan*
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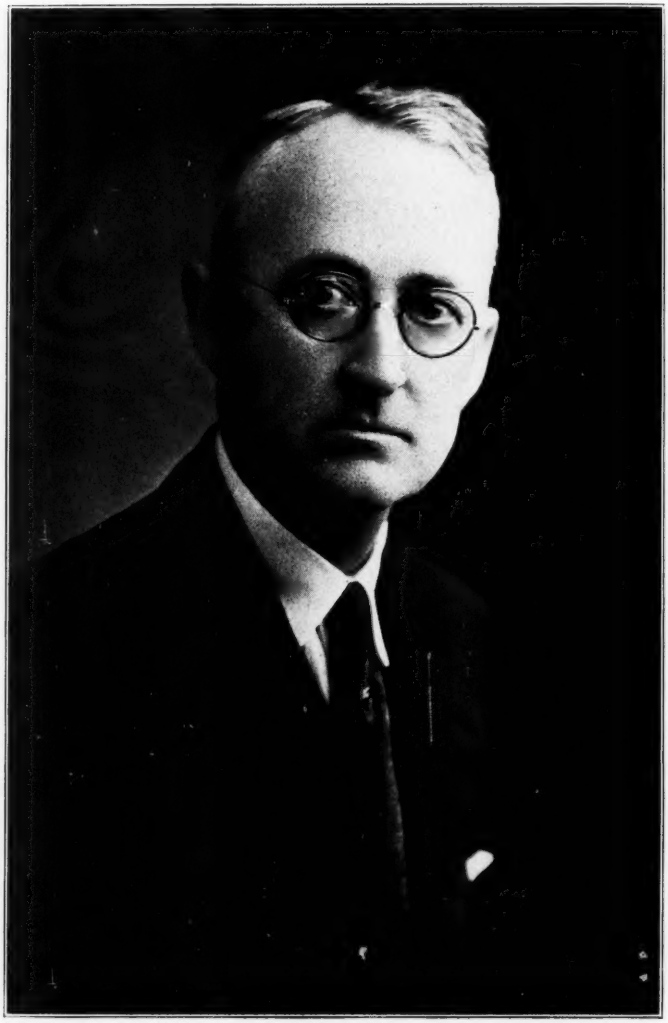

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UNIV. OF MICH.

THE JOURNAL
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## Original Communications

# HORNIFICATION OF THE GUMS*

By BALINT ORBAN, M.D., Vienna, Austria

DENTISTS have at all times to deal with living tissues. This is true if we prepare a cavity in the dentin or if we make a denture which is to be supported by the tissues of the jaw. But it is more evidently true when we have to deal with gum conditions. We believe that it is just as important to watch the condition of the gums of our patients as it is to examine the teeth for cavities every six months. Especially does this hold true for individuals who are inclined to have calculus deposits and who do not believe it is necessary to massage the gums twice a day. We must also know what happens to the gums if we neglect them and we must know how they react if we take care of them. It was for this reason that the following study was started.

There is a thesis that normally the mucous membrane of the human mouth does not become hornified. The literature on the hornifying process of the skin is voluminous. Concerning this change in the mucous membranes, there is less to be found, yet important investigations regarding it have been made. Severin, who has made comparative examinations on the hornification of mucous membranes, comes to the conclusion that the oral mucous membrane, both in human beings and in certain animals, shows hornification only in certain parts of the tongue. He was able to demonstrate keratohyalin granules.

These statements were confirmed by Posner. Both authors have considered the occurrence of keratohyalin granules, which exist in the form of a granular layer in the epithelium, as a sign of genuine hornification. Posner writes: "One should always bear in mind this strange substance, which lately under the

*From the Histologic Laboratory (Director, Bernhard Gottlieb) of the Dental School of the University of Vienna (Chief, Hans Pichler).

name of keratohyalin or eleidin, has taken such a large part in the histology of the skin." Both authors, Severin and Posner, found that the horny layer of the mucous membrane of the mouth was not free of nuclei, but, as Posner writes, "shows pale, thin nuclei." He attributes this to the fact that the mucous membrane is constantly moistened.

V. von Ebner writes, in Koelliker's "Handbook of Tissues":

> With the exception of the epithelia of the lip and a part of the tongue, the human mouth epithelium shows no signs of hornification; but this, to a greater or less degree, is present in animals (tongue of the cat, palate of the ruminants, etc.). Thus, one finds also in animals a layer of epithelium similar to the granular layer of the epidermis in which the cells contain keratohyalin or eleidin granules. Severin found the same in human beings, cattle, pigs, rabbits, rats, dogs, cats and moles.

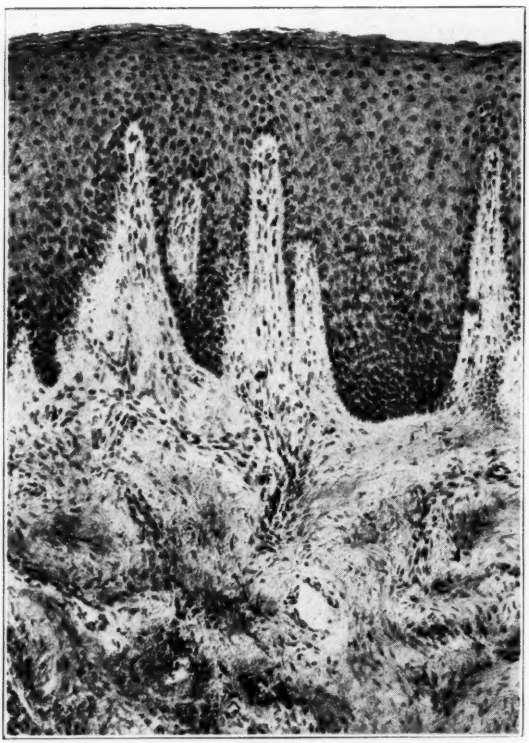

Fig. 1.—Interdental papilla of an individual aged 31. The gums looked normal clinically, were pale pink and surrounded the teeth firmly. Mouth hygiene was good. (× 100.)

> Up to now, in adult human beings, I could find keratohyalin granules only in the region of the filiform papillae of the tongue.

V. von Ebner writes again: "The cells contain nuclei throughout. Their arrangement is very similar to that of the

epidermis, but normally does not reach hornification, with the exception of the edge of the lip, and the cells of the deeper layers gradually merge into the platelets of the surface."

We find descriptions in the dental literature which correspond to these statements. Siegmund and Weber, in their book on pathologic histology of the oral cavity, write that real hornification of the mucous membrane of the mouth does not occur with the exception of the filiform papillae of the tongue, but that keratohyalin formation is found there. Otherwise, "under normal circumstances, only a very thin superficial layer of parakeratotic horn is found in the oral mucous membrane." They saw genuine hornification only in association with pathologic conditions, such as leukoplakia or black hairy tongue.

Praeger writes, in his treatise on "The Gum Margins": "The opinions in the literature are divergent as to the extent to which genuine hornification comes into consideration. To avoid confusion this layer should be called the 'wearing layer.'" Schumacher asserts, in his contribution, "The Mouth Cavity," in Möllendorff's handbook: "The epithelium shows the typical arrangement of the stratified squamous epithelium with its various cell forms, which are becoming flattened as they approach the surface. It is distinguished from the epidermis, especially by the fact that, in human beings at least, there is not, as a rule, any hornification in the superficial cell layers, so that the superficial flat cell layers also contain granules."

Recently, Bottler has taken up this question and he states that the mucous

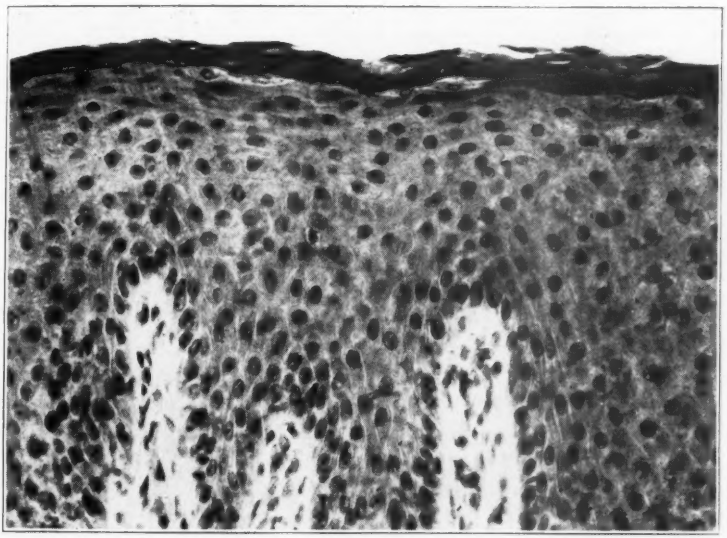

Fig. 2.—Higher magnification of Figure 1. Normal epithelium with no wandering cells in the intercellular spaces. A thick layer of horn covers the outer surface. (× 270.)

membrane of the mouth is distinguished from the skin of the body by the fact that keratinization in the mouth is to be considered as pathologic, whereas the same process in the skin is physiologic. Parakeratosis, which occurs in the skin only under pathologic conditions, is to be considered as a normal condition of the oral mucous membrane. Bottler seems to attribute this difference to the moist condition of the mouth as opposed to the dryness of the skin.

gingiva into consideration. Siegmund and Weber deal with the oral mucous membrane in general, and though they ordinarily deal with questions which are of interest to dentists, they failed to examine the gums. Bottler did likewise; in fact the only one who examined even portions of the gums was Praeger, but he did not come to a decision with regard to the question of hornification.

In the other dental literature, hornification of the gingiva has often been men-

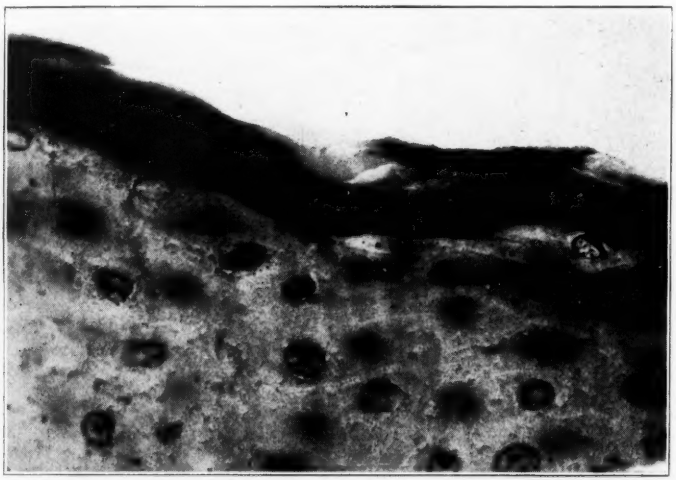

Fig. 3.—Higher magnification of Figure 2. The horny layer, which is sharply separated from the epithelium beneath, contains darkly stained, flattened nuclei. The cells of the epithelium show small dark granules, the so-called keratohyalin granules. The nuclei of these cells show a defective staining of the chromatin. (× 800.)

What I wish to point out to these research workers is that they are generalizing. What is normal for the cheek must not necessarily be normal for the tongue, the lip, the palate, the gingiva propria or the gum margins. Severin, and also Ebner, though they have examined different parts of the mucous membrane of the mouth, have not taken the

tioned, but whether the condition was considered to be keratosis or parakeratosis was not discussed. Gottlieb considers hornification of the epithelium of the gums to be the ideal condition.

The "gingiva propria" is the part of the mucous membrane which surrounds the teeth and forms the gums and the interdental papillae. This portion of the

gingiva is bound fast to its under layer because it possesses no loose submucosa. When in a healthy condition, the gingiva propria is a pale pink. It is bordered by an undulating line toward the peripherally situated mucous membrane. The "gingiva mucosa" which, on account of its loose submucosa, can be easily removed, is very delicate, and, because of its richness in blood vessels, is dark red.

In order to study the inflammatory conditions of the gum, I have taken some gum papillae from individuals between 15 and 60 years of age. These portions of tissue were immediately put in a fresh fixation fluid previously prepared. I used chiefly a Zenker-formol mixture, recommended by Maximow, or absolute alcohol, and 5 per cent neutral formalin. I must point out particularly that the gum portions were placed in the fixation fluid immediately after their removal, so that there was no possibility of their drying out. The tissues were fixed for from three to ten hours and then embedded in the usual way in celloidin. For the study of the hornification, the stain used was hemalum-eosin. For the investigations on inflammation, chiefly hematoxylin-azure II-eosin and other special stains were used. For the identification of hornification, Gram-Ernst's dye was used, and, according to Unna's directions, incubator digestion in hydrochloric acid and pepsin was carried out.

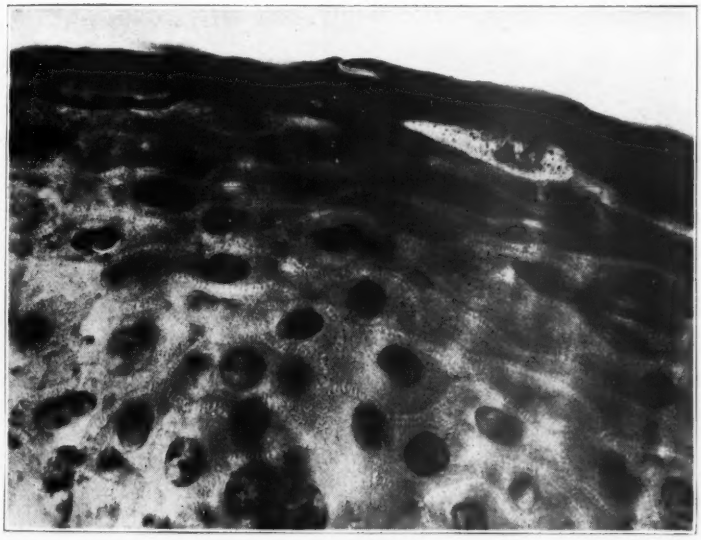

Fig. 4.—Specimen shown in Figures 1-3, but from a different region of the section. The granular layer of the epithelium (stratum granulosum) is distinctly separated from the superficial horny layer. The prickle cell layer (stratum spinosum) is characterized by the intercellular bridges. The horny layer (stratum corneum) shows only a few remnants of cell nuclei, being for the most part free of them. (× 800.)

In the following report, I wish especially to deal with the hornification process of the gingiva propria and of the gums.

Immediately, in the beginning, the difficulty arose as to what one should regard as normal gums. It must be taken into consideration that because of the relationship of epithelium and calcified hard tissue, special conditions arise which have no analogy in the remainder of the organism. The epithelium of the gingiva propria forms a band on the surface of the teeth (the epithelial attachment of Gottlieb). Thus, the gum margin and is a crevice which reaches the edge of the epithelium and is called the gingival pocket or gingival crevice. This is a hiding place for bacteria and is certainly in other respects as well the most unclean part of the mouth, because both soft and hard deposits from the saliva and small portions of food collect and remain here. Thus mechanical, chemical and bacterial irritation to the gingiva results. It is to these circumstances that the usual chronic

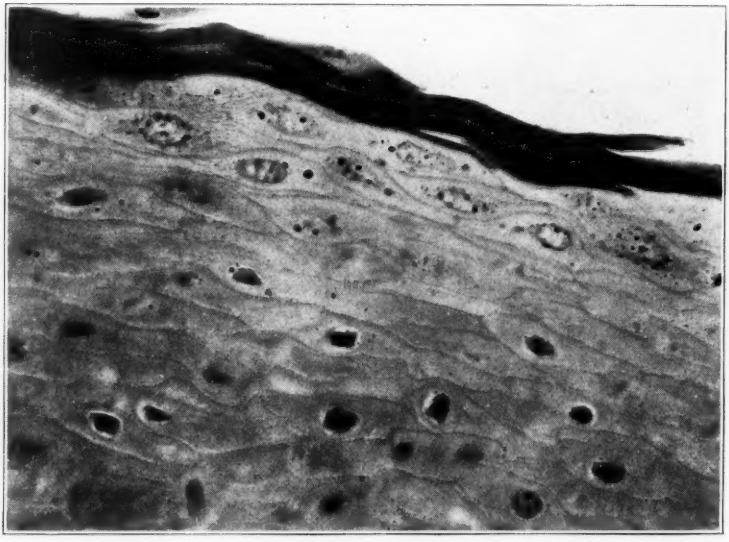

Fig. 5.—Gum of an individual aged 32. The gums showed a somewhat reddish border, but did not bleed and the patient used the toothbrush in a proper manner. Microscopically, inflammatory cell reaction was seen only in the deeper regions of the connective tissue. The horny layer shows a lamellated structure and nuclear cavities. Only a few flat nuclei are present. The cells under the horny layer are filled with keratohyalin granules. (× 600.)

the interdental papillae are formed. In this relationship of mucous membrane and hard tissue, there is a certain danger for the mucous membrane because the tissue is constantly exposed to the mechanical act of chewing. Between the tooth surface and the gum margin, there inflammatory condition of the gum border is due. The small cell infiltration of the marginal portion which was believed to be constantly present has already been described as being physiologic (Ebner), but it could be shown that gum margins free from inflammation are sometimes

seen (Gottlieb), and these are considered to be ideal. In the material that I have collected, I was able also to observe gum papillae free from inflammation, though in the portions near the gingival crevice I was able to find signs of inflammatory reaction in a greater or less degree in most of my specimens.

Figure 1 shows a section of the interdental papilla of an individual aged 31. Clinically, the whole gingiva propria, with the gum margin and the interdental papillae, was an even pale pink. The gums, which lay firmly around the teeth, could be described as normal. For cleaning the mouth, a hard toothbrush had been used and with this the border of the gums had also been brushed according to the method of Charters combined with that of Stillman. The epithelium presented a uniform condition, narrow intercellular spaces with intercellular bridges being present throughout. There were no wandering cells in the epithelium. The connective tissue papillae of the tunica propria reached upward toward the surface to about the tenth row of epithelial cells. The epithelial processes show no signs of growths; the subepithelial connective tissue is free from inflammatory infiltration, and therefore the gum can also be considered normal microscopically. Already, at this magnification, the superficial layer of the epithelium is obviously differentiated from its under layer. In this specimen, the superficial layer is stained a brilliant yellowish red with eosin and is distinguished therefore by oxyphilia.

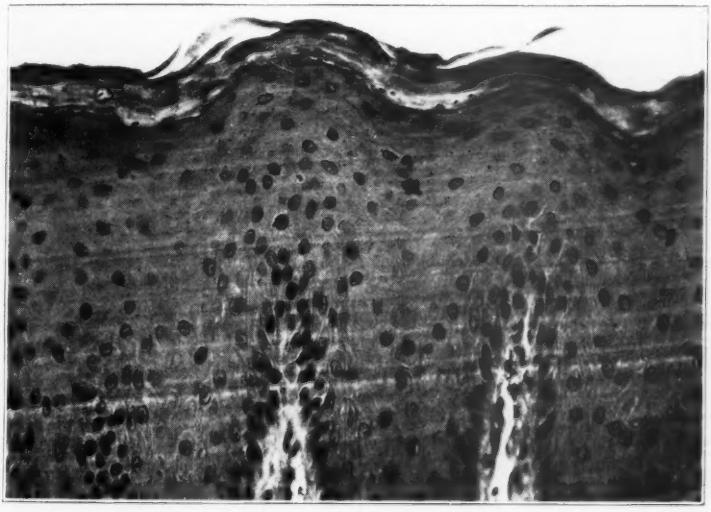

Fig. 6.—Interdental papilla of an individual aged 24. The gums looked clinically healthy, in good condition and well cared for hygienically. The wavy outer surface of the gum is covered with a heavy horny layer. (× 270.)

In a higher magnification of this portion (Fig. 2), we can see the cells of the *stratum germinativum et spinosum.* (I

cannot go into details and present arguments regarding the classification of the different layers of the epithelium, but I call attention especially to the treatise by Hoepke [1927] in Möllendorff's handbook on "The Skin.") Toward the surface, the cells of the prickle cell layer (stratum spinosum) become flatter, and are elongated. This change of form of the cells from the basal cell layer toward the surface is, according to Heidenhain, due to differences of tension.

fied, i. e., parakeratotic, some cells with granules, stained blue in the specimen, are seen. The nuclei of some of these cells show incomplete staining of the chromatin. This layer we may call stratum granulosum. The cells contain keratohyalin granules. A stratum lucidum cannot be distinguished. We can see some platelets being cast off from the surface (stratum disjunctum).

Another portion of the same specimen in the same section shows a somewhat

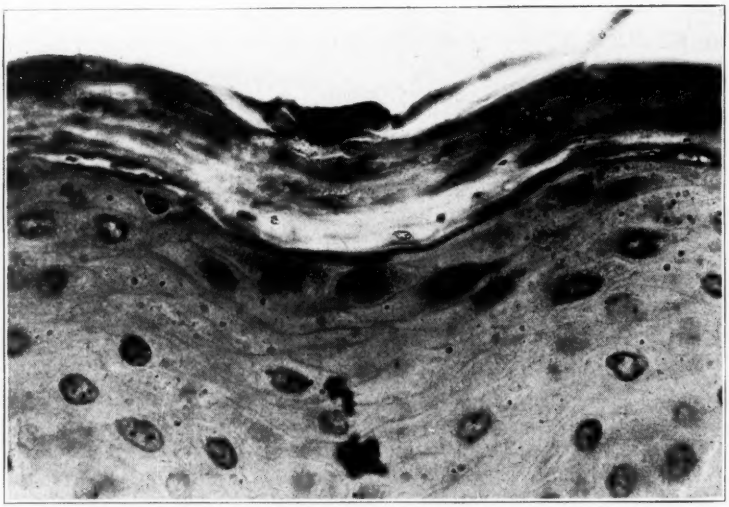

Fig. 7.—Higher magnification of Figure 6. The horny layer has no nuclei, and the cells under this layer show keratohyalin granules. (× 720.)

Figure 3 shows a still higher magnification of the superficial layer of the epithelium. The uppermost layer looks dark in the photograph, but in the microscopic section, as already mentioned, it is a brilliant yellowish red. Elongated cell borders and flat, narrow and very dark colored nuclei are to be recognized in this layer. Underneath this layer, which may be recognized as incompletely horni-

different condition. (Fig. 4.) We see here the prickle cell layer flattening toward the surface and the color of the nuclei becoming indistinct. Under the stratum corneum are elongated cells, rather pale, and filled with keratohyalin granules. The nuclei of these cells show incomplete chromatin staining and in them may be seen "the signs of breaking down," as Hoepke expresses it. The

horny layer, which is bordered by a well differentiated line and extends toward the periphery, is called the stratum corneum. As opposed to the former picture, we see here, quite close to the border line, some flat cell nuclei, but otherwise the layer is free of nuclei. On the surface, scales are being thrown off.

We can describe this as a genuine hornification with production of keratohyalin granules and a stratum corneum free of nuclei.

I should mention here that this stratum corneum appeared blue when stained with Gram-Ernst dye and that it was resistant to digestion.

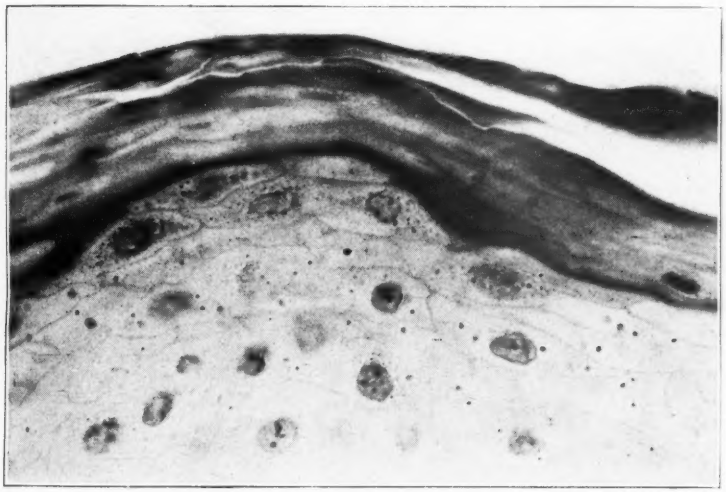

Fig. 8.—Specimen shown in Figures 6-7; a heavy horny layer, with a few nuclear remnants. The upper layers of the horny layer are being cast off or shed. Keratohyalin granules are present in the epithelium. (× 720.)

It is, of course, not my aim to discuss the origin and importance of the keratohyalin, but I draw attention to contributions on this subject, mentioning especially those by Biedermann, Hoepke, Koelliker, Kromayer, Kyrle, Patzelt, Rabl, Unna and Weidenreich. From the study of this literature, it is clear that an agreement about this and many other questions concerning the stages of hornification could not be reached.

Some research workers believe that keratohyalin is a product of the cell nuclei; others, that it is proved to be a product of epithelial fibers or that it is produced by a change in the plasma or the breaking down of the interfibrillar substance. Variations of these theories have been advanced, and even the possibility of the origin of the keratohyalin and its constituents from the blood and lymph has been discussed.

No conclusion has been reached regarding the relationship of keratohyalin to hornification and how the biologic process of hornification takes place.

It is certain that keratin is not a product of keratohyalin. Unna showed

that, by digestion, keratohyalin disappeared; whereas, keratin did not. He has expressed his views as follows: "The hornifying epithelia mostly produce keratohyalin, but, in no instance, is the horn substance of the horny layer formed by the keratohyalin of the granular layer, because both substances can be found in different places inside the same cells."

It has been stated that the cells first hornify on the periphery; whereas, the keratohyalin occurs in the protoplasm itself. Unna explains the formation of the keratohyalin by saying, "It is biologically the most simple when it is considered to be a resulting condition of the general *oxygen loss* which introduces the beginning of each process of hornification."

Kromayer found that the keratohyalin containing cells are almost as much hornified as the real horn cells. He believes that the hornification takes place independently of the keratohyalin. "If there were any relationship, we could only imagine that it exists in this way—that the keratohyalin is the result of hornification."

Kromayer considers the hornification process to be a necrobiosis of the epithelial cells. "The keratohyalin is the histologic expression of a necrobiosis of the epithelial cells." He thus explains the change in the cell nuclei also.

He says further: "The hornification process is therefore gradual and uniform, progressing throughout the entire thickness of the epidermis on to the edge of

Fig. 9.—Gum of the individual referred to in Figures 6-8; six months before the specimen in Figure 6 was taken; i.e., before any treatment was started. The gums looked red and swollen and bled easily. The "horny cells" have mostly no nuclei or only remnants of them. The granular layer is very plainly to be seen. Owing to shedding on the outer surface, the horny platelets are loose. (× 720.)

the real horn layer. This process consists of a thickening and solidifying of the cell membranes."

Koelliker also holds the view that the granules of the stratum granulosum are not necessarily precedents of hornification. Weidenreich upholds Unna's view that the stratum corneum is built up with the same material throughout, right up to the surface, and this Unna proved by his digestion experiments. These experiments have also shown that keratin exists, keratin inside which nuclear remains may sometimes be seen in certain fixations. Whereas the content is diluted in the digestive fluid, the membrane is indigestible." Hoepke does not consider the hornification process to be entirely a degenerative one because important biologic changes take place in it. He considers the occurrence of keratohyalin granules and the membrane formation of the cells to be the beginning of hornification. Patzelt comes to the conclusion that

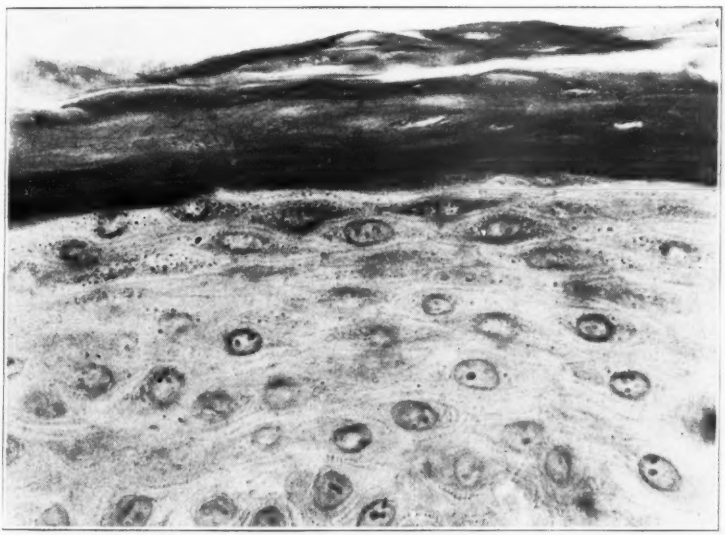

Fig. 10.—Specimen shown in Figure 9, from another region. There is a sharp line between the granular layer and horny layer. (× 720.)

not inside the cells, but only on the surface, in the form of a thickened membrane. Inside this horn membrane is the so-called nuclear cavity. Hoepke believes that this cavity is not hollow, "but filled with a substance which is difficult to stain." He writes, "The cells of the horn layer are flat scales plastered on one another with a membrane consisting of keratohyalin "has no relationship to keratin but occurs only in hornification processes."

The possibility that the drying up of the cells has some influence has also been taken into consideration, and authors of prominence believe that, in the process of hornification, there is a drying process. Against this view is the fact that the

epidermis also hornifies in animals which live in the water, and that hornification also occurs even in intrauterine life. The constant moistening of the mucous membranes was taken to be the explanation for their incomplete hornification.

I will leave until later the decision as to how much my findings speak for the one theory or the other. The findings themselves are herewith stated:

Figure 5 shows the stratum granulosum and stratum corneum of an individual aged 32. The hygienic conditions of the mouth were good. The patient has used the toothbrush for many years as we teach its use. The gums showed rather reddened borders macroscopically, but microscopically only a very little infiltration could be observed. The cells of the stratum spinosum are more and more flattened toward the surface and, in the upper three or four cell rows, keratohyalin granules occur in the cell body. The nuclei of these cells simultaneously

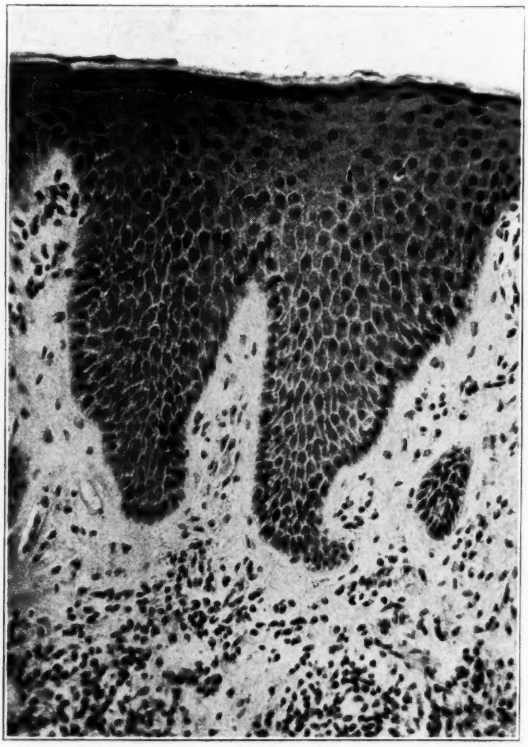

Fig. 11.—Gum of an individual aged 36. Clinically, signs of inflammation were present. This was confirmed microscopically. The inflammation is in the deeper regions of the connective tissue. The epithelium is fairly near the normal, showing a few wandering cells. The outer surface is shown in Figure 12. (× 170.)

lose the normal coloring power of their chromatin and become pale. Intercellular bridges are also to be seen between the cells of the stratum granulosum. This layer is differentiated from the stratum corneum by a distinct border, the latter stratum being built up in lamellae and showing a few dark colored longitudinal nuclei or their remains. On the surface, some scales are being thrown off. Light spaces in the horn lamellae correspond to

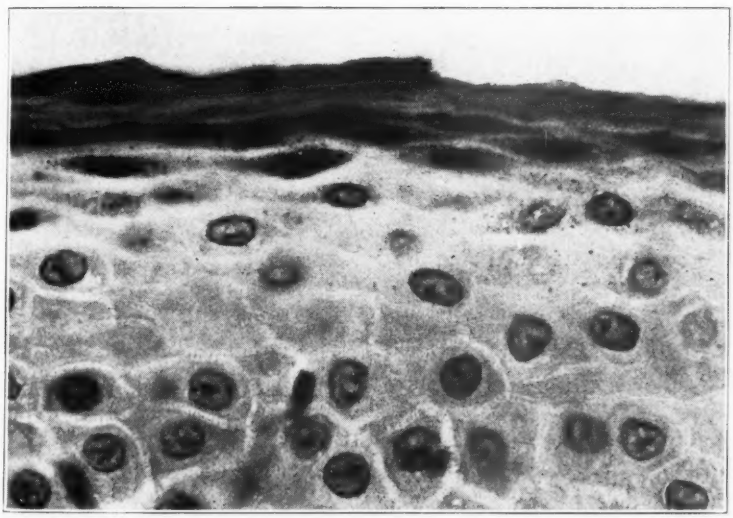

Fig. 12.—Higher magnification of Figure 11. The horny layer is free of nuclei. The cells of the granular layer are filled with keratohyalin granules. (× 800.)

the conditions which are described in the literature as nuclear cavities.

From the same individual, a portion of the gums was taken at various times, and each time a horn layer and keratohyalin production could be demonstrated. Not all the horn layer showed the same consistency, but portions which were completely free of nuclei were interspersed with others that contained longitudinal dark nuclei or their remains.

Figure 6 is a section taken from an individual aged 24, who, six months before the removal of the gingival portion, came in for treatment with livid and swollen gums, bleeding at the pockets and much tartar present. After efficient cleaning, the patient was taught how to use a toothbrush in a rational way. When the gum papilla was removed and photographed (Fig. 6), the gums looked normal, healthy, pale pink and firm, and surrounded the teeth firmly. Bleeding of the gums had stopped completely.

On the undulating surface of the epithelium, a strong horn layer, completely free of nuclei, is seen. A granular layer can be found in the troughs of the waves (Fig. 7) as well as on the

crests (Fig. 8). The cells in the stratum granulosum are crowded with keratohyalin granules. In Figure 8, there are few remains of nuclei in the stratum corneum, but this layer is mostly free of nuclei.

After the description of these three cases, one might be led to believe that the marked hornification of the gums might have been caused only by intensive brushing and thus be due to mechanical stimulation. That this is not entirely the case may be seen in the following pictures.

Figure 9 was taken from the gum of the same individual as Figure 6, but before the treatment was commenced. The gums were, as previously mentioned, livid, swollen and bleeding from the pockets, where much tartar was present. Because of the bleeding, the patient had not brushed the teeth for some time. A considerable horn layer is seen here, lying on the epithelial surface. The stratum granulosum consists of from three to four cell rows. Between some cells, we see horn lamellae protruding and surround-

Fig. 13.—Gum of an individual aged 22. The gums were red and swollen and bled very easily. Microscopically, an irregular epithelium and a heavy subepithelial inflammatory cell infiltration are seen. A thick outer layer covers the epithelium. (× 100.)

ing the cells filled with keratohyalin. These might possibly be horn membranes of neighboring cells and so this "surrounding" would be only a condition caused by the direction of cutting the section. It is also possible that some cells are hornifying here at the periphery; whereas, inside the cells, keratohyalin is still present. Empty nuclear cavities or breaking down nuclear remains may be found in the horn lamellae.

crowded with keratohyalin granules and the nuclei are breaking down. The stratum corneum is definitely distinguished from the cell layer. Nuclear remains can be seen only in the innermost part of the horn layers. Otherwise, the "horn cells" show only "empty" nuclear cavities. The subepithelial connective tissue showed signs of chronic inflammation, not in the stratum papillare, but in the deeper layers.

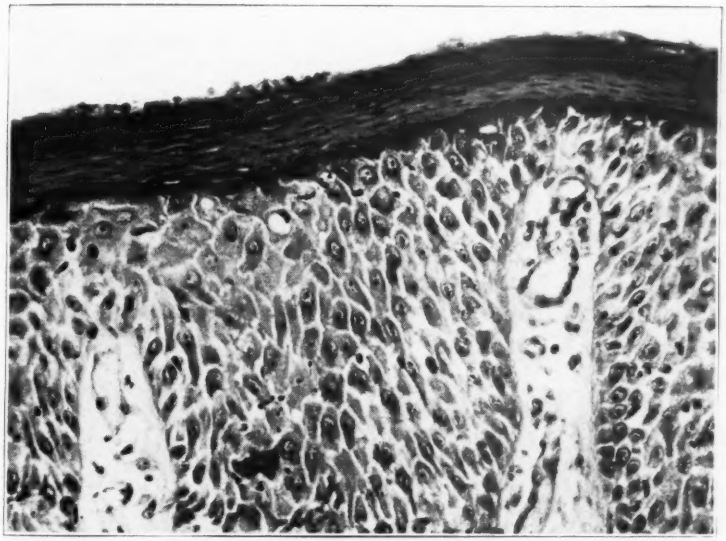

Fig. 14.—Higher magnification of the outer surface of the epithelium in Figure 13. The outer layer of the epithelium shows a large number of small, flat, darkly stained nuclei. This layer has undergone "parakeratotic" hornification. The horny layer is sharply separated from the cells of the epithelium, which are not flattened and do not contain any keratohyalin granules. (× 270.)

The specimen shown in Figure 10 is from the same subject and taken at the same time as the one which has been previously described. The stratum granulosum has about five cell rows. The cells in this layer are flattened and spindle shaped. The body of the cells is

Whether a hornification of the gums will be found everywhere that healthy conditions are present and there is rational hygiene of the mouth, or whether it occurs only in certain predisposed individuals who may or may not look after their mouths well, is something that

requires further examination and this examination is under way. According to my experience, I dare to make the statement that by correct mouth hygiene, hornification of the gingiva propria can be obtained in every case. In some cases, it will be less, in others more marked, varying with the disposition toward hornification in general. That such a disposition seems to exist may be seen in our case in the difference of the process in different individuals during healthy and diseased states.

A further case of normal hornification is shown in Figure 11, taken from an individual aged 36. The gums showed clinical signs of inflammation, which was also expressed microscopically by infiltrated areas of the connective tissue. The stratum papillare of the tunica propria did not seem to be much affected by the inflammation. The infiltration is found chiefly in the area corresponding to the stratum reticulare and is concerned mainly with plasma cells. In spite of the inflammation, the epithelium is in a good condition. Only a few signs of growth and some wandering cells in the intercellular spaces may be observed.

A further enlargement of the stratum granulosum and stratum corneum is reproduced in Figure 12. The horn layer, completely devoid of nuclei, is sharply distinguished from the cells filled with keratohyalin granules of the stratum granulosum. The lamellae-built stratum corneum shows numerous nuclear cavities.

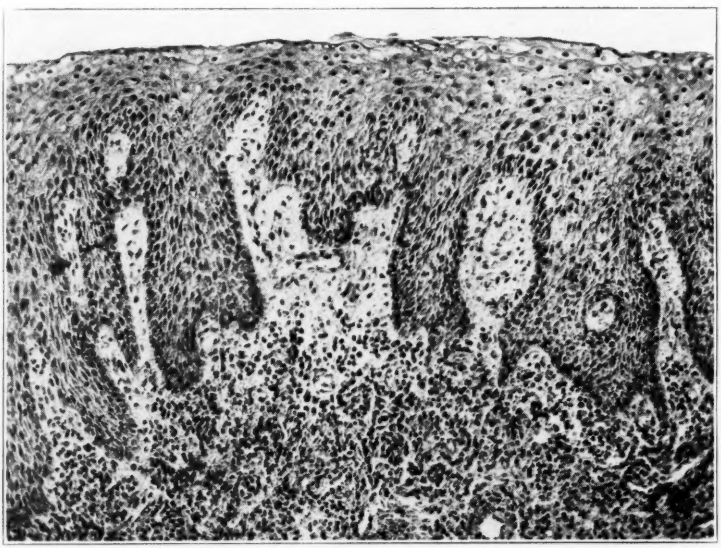

Fig. 15.—Gum of an individual aged 56. The gums were red and bleeding. There is irregular epithelial proliferation into the connective tissue, which is heavily infiltrated by inflammatory cells. The upper layers of the epithelium show vacuoles and no hornified layer covers the outer surface. (× 100.)

I believe that I have demonstrated from the foregoing paragraphs that it is not justifiable to say that mouth cavity epithelium in general is not normally hornified. This might be correct for the cheek, the floor of the mouth, etc., but it cannot be applied to the gingiva propria, the gum border or the interdental papillae. These normally show genuine hornification. That this hornification cannot always be found may be due to the especially close relationship of the gums to the teeth and to their accompanying influence. Individual disposition might also play a part in this.

If I have been successful in showing that the gums normally undergo genuine hornification, I should be allowed to say that parakeratosis is a pathologic condition of the gums. Parakeratotic hornification is described in special textbooks as a process of incomplete hornification. It shows no keratohyalin granules during formation and nuclei in the horny layer. Genuine hornification shows keratohyalin granules in the cells below the horny layer and no cell nuclei in the horn.

The gums microscopically reproduced in section in Figure 13 showed the clinical picture of an acute gingivitis, in a 22-year-old patient. The gum margins were characteristically very red and bled when touched gently. The epithelium here has an irregular appearance and the epithelial processes penetrate irregularly into the tunica propria, the stratum papillare being irregular and reaching almost to the surface of the epithelium. In some parts of the specimen, the papillae are found to extend to immediately beneath the parakeratotic surface layer. The papillae as well as the deeper layers of

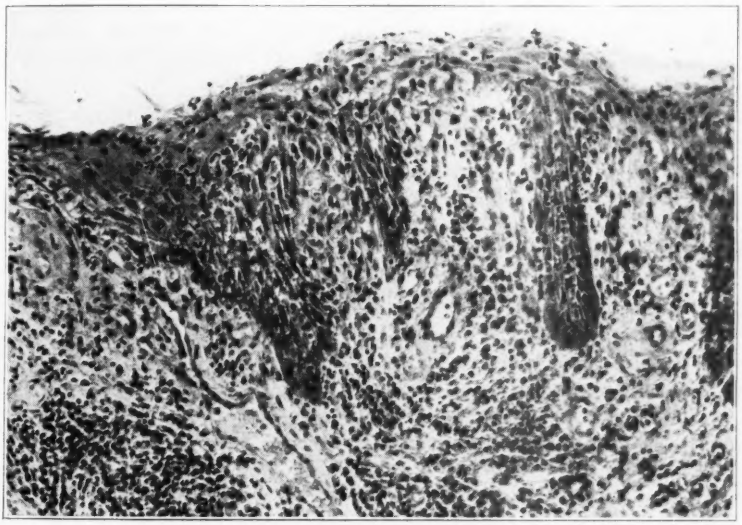

Fig. 16.—Case shown in Figure 15. Leukocytic wandering cells invaded the entire epithelium. There is no hornified layer covering the outer surface. (× 100.)

the tunica propria are infiltrated. Plasma cells, lymphocytes, polyblasts and polymorphonuclear leukocytes are infiltrated.

We see in higher magnification (Fig. 14) that the intercellular spaces are considerably enlarged and that the cells of the stratum spinosum show signs of degeneration. The surface layer is sharply distinguished from the cells of the stratum spinosum without the latter's being flattened on this border line. Instead of the stratum corneum without nuclei, we find nuclei-containing structures. The nuclei are flat, longitudinal and dark. As we have here the typical picture of parakeratosis, we consider that we are justified in comparing it with a picture of psoriasis of the skin. It represents, in its own way, a very extreme case.

The discussion of two more pictures, from the same individual, aged 56, should close our study. Figure 15 shows the interdental papilla, with very irregular epithelial proliferation. The processes of the epithelium are different in size and form. This epithelium should be compared with that in Figure 1, which is absolutely normal. The epithelium in Figure 15 appears to be degenerated. Many vacuoles are to be seen, especially in the upper layers. No hornified layer covers the epithelium. The degenerated cells of the stratum spinosum build its outer layer. The nuclei of these cells seem to stain pale, which is a sign of degeneration.

In the subepithelial connective tissue, large masses of inflammatory cells are gathered, consisting mainly of plasma cells and polyblasts.

Clinically, the gum in this case was swollen and reddish blue, as is usually seen if circulation in the tissue is not satisfactory.

Figure 16 is from the same case, nearer the gingival crevice, where, owing to the irritation, the inflammatory reaction is more acute. Large masses of leukocytes are wandering through the epithelial layer, between the cells, to the surface. The epithelium is thin and irregular. The cells are mostly in a stage of degeneration. The intercellular spaces are wide and filled with wandering cells. The connective tissue shows the same cellular elements as the specimen shown before, but in addition a large number of leukocytes are present—the characteristic sign of acute inflammation. No hornified layer is to be seen on the surface of the epithelium.

We have seen clinically as well as microscopically different pictures. With the exception of the last two, which showed no hornification whatever, the pictures showed different degrees of it. The last two pictures are from a case showing severe inflammation, and we may suppose that the inflammation is to be held accountable for the loss of the hornified layer. In some other cases of inflammation, we find different degrees of hornification, as in Figures 13-14 (parakeratosis), or in Figures 11-12 (normal genuine hornification).

In all the cases in which clinically the gum was healthy and it proved to be so in the microscopic examination, we found more or less evident genuine hornification with production of keratohyalin granules and a horn layer without cell nuclei. Therefore, we have to consider genuine hornification as the normal and ideal, and should attempt to produce it by our prophylactic measures, i.e., scaling and teaching the patient a rational toothbrushing method, with massage of the gums. A normal and properly hornified gum is firm and pale pink, and tightly

attached to the teeth, and does not bleed under common mechanical insults.

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# THE CASE AGAINST TRAUMATIC OCCLUSION*

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"PREJUDICE is a defense reaction of ignorance. You're apt to be 'down on' what you're not 'up on'."[1]

With this quotation in mind throughout the paper, a number of apparent discrepancies in the "doctrine" of traumatic occlusion will be presented in the hope that they may stimulate a constructive discussion and that "we shall try honestly to understand each other."[2]

"Traumatic occlusion" is defined as "an abnormal occlusal stress which is capable of producing or has produced an injury to the periodontium."[3] "Briefly stated, it consists of an application of occlusal force in excess of that which the tooth is capable of withstanding."[4] " . . . traumatic occlusion is the predominating factor in the etiology of a large percentage of the cases of suppurative periodontal disorders."[5]

"Traumatic occlusion is an abnormal stress." The term "abnormal stress" is used with as much assurance as though the actual number of pounds pressure of overstrain had been definitely determined. Since the maximum amount of stress that the periodontium is able to stand without injury has never been ascertained, how can the term "abnormal" be used in the definition which "should fix or prescribe clearly and authoritatively"? The recognition of the fact that a certain amount of stress must be present for the normal development of the jaw and for maintenance of its resistance to disease in the years after development is of paramount importance. Normal adult form cannot be attained without the stimulation of internal stress. How much stress? The strategic point at which normal stimulation ends and retrogressive changes are claimed to begin as the result of masticatory overstrain has not been determined. Until this strategic point has been found, the term "abnormal occlusal stress" is of no scientific significance.

"Capable of producing" is an interesting phrase for analysis. Traumatic occlusion is claimed to be an abnormal stress which "possesses the power or capacity of producing an injury." The word "capable" implies the power but not the performance. In other words, there are, according to the definition, cases of abnormal stress which are capable of producing but do not produce an injury. The inference that "abnormal stress"

*Read before the Section on Periodontia at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 22, 1930.

1. Overstreet, H. A.: Influencing Human Behavior, New York: W. W. Norton Co., Inc., p. 248.

2. Footnote 1, p. 254.

3. Stillman, P. R., and McCall, J. O.: Textbook of Clinical Periodontia, New York: The Macmillan Co., 1922, p. 63.

4. Footnote 3, p. 52.

5. McCall, J. O.: Principles of Traumatic Occlusion and Their Application in the Field of Operative Dentistry, J.A.D.A., 17:609 (April) 1930.

may at times produce an injury *under certain conditions* discloses the weakest part of the argument and places the chief emphasis on the "certain conditions." "Capable of" in the definition implies doubt that traumatic occlusion is always the cause of pyorrhea alveolaris, and stresses, inadvertently, that, at times, it is not.

The last part of the definition, "or has produced an injury," is an assertion too conclusive. "Traumatic occlusion" was first introduced as an hypothesis; it has never advanced to the dignity of a theory; it is too often accepted by undiscriminating dentists as a fact. According to the published writings of McCall, alveolar border disturbance has never been produced experimentally by placing "a perfectly healthy tooth" in "traumatic occlusion."[6] Since the definition is not founded on experimental methods, it must have originated from observations. Is it not possible that clinical findings have been misinterpreted?

In consideration of the statement, "Briefly stated, it consists of an application of occlusal force in excess of that which the tooth is capable of withstanding," the following quotation from Sir Arthur Keith is quite apropos:

> Every one of us has been given the appetite, not of a brain worker but a muscle user. You may study to the utmost limit of your endurance and by the mere act of study you may rest assured you will do your brain no injury. By habitual study you may damage your body if you neglect exercise, without a doubt; but you certainly will not damage your brain. All our organs are built for emergencies at critical junctures. *The heart can rise to ten times its normal output.* So in the same way can the brain.[7]

By analogy, may it not be assumed that the periodontium may resist, in emergencies, ten times its average stress without injury?

In the "Textbook of Clinical Periodontia," there are a number of statements which have never been qualified:

> When a condition of plus occlusion is found to be present, one of two things will occur; either Nature will provide an increased alveolar support . . . or a breaking down of the supporting tissues will take place.[3]

The fact that "Nature will provide an increased alveolar support" has been demonstrated, proved and accepted. The latter part of the quotation is an unsupported statement which has not been demonstrated, according to McCall, by experimental work deliberately planned "in order that we may round out our story."[6] The mere recital of the classic microscopic changes that occur in inflammation is not sufficient. It has been demonstrated that "breaking down" is due to disuse—not overuse. The claim that two opposite causes produce the same pathologic condition makes a travesty of dental science. One or the other is in error.

> Unfortunately, the tendencies of civilization are toward more frail alveolar structures, the consequences being a corresponding tendency toward degeneration.[8]

This statement introduces the following argument which one sees frequently in dental literature: "Since the alveolar process is not developed as in primitive man, it is not able to withstand the forces applied to it through the muscles of mastication." It is an interesting observation in going over the literature on "traumatic occlusion" that the origin of the "tremendous amount of overstrain" is scarcely considered. As a matter of fact, after eruption, the teeth *per se* are inca-

6. McCall, J. O.: Roentgenogram as a Diagnostic Aid in Planning Partial Restorations, J.A.D.A., 15:1003 (June) 1928.

7. Keith, Arthur: Pharmical Advance, Vol. 8, No. 96, 1929, p. 4.

8. Footnote 3, p. 64.

pable of exerting an ounce of force. The alveolar processes and jaw bones are under no resistive stresses when idle. The whole mechanism of stress and strain begins when the muscles attached to the mandible begin to contract. Has this degenerative process which has produced the "frail alveolar process" no effect on the structure of the muscles? Has present day civilized man the same degree of muscular development as primitive man? Is the present day "frail" mandible forced to resist the leverage of muscles which are as powerful as the muscles of primitive man? Paraphrasing the former statement: Is it not equally true that, unfortunately, the tendencies of civilization are toward more frail muscles of mastication as well as more frail alveolar structures? Mackenzie states: "Bone is dominated by muscular function, for that it is called into being, with the disappearance of that necessity, so also disappears the bone."[9] "It is the length, size and 'tonicity' of the muscle that maintains the normal length and size of the bones, and not the reverse, as is so often erroneously taught."[10]

The "overstrain" claimed to be present on "frail alveolar structures" must be produced by correspondingly frail muscles of mastication.

The statement has been made before and is still unchallenged that "in the roentgen ray negatives used to describe occlusal traumatism . . . evidence of disuse atrophy, the antithesis of functional stimulation, is more often present."[11] The appearance of the external contour and internal architecture of bone is a criterion of the amount of muscular force resisted. A "weak frail alveolar structure" is the result of insufficient stimulation of muscles also affected by disuse atrophy.

A study of "overstrain" and "occlusion plus" should start at the source of power. There is no report, in the literature, of the gnathodynamometric readings of the muscles of mastication which produced, by their excessive power, the beginnings of pyorrhea alveolaris.

As to the amount of force exerted, it is important to realize that the hundreds of pounds pressure which may be exerted by well developed muscles during incising or masticating is present only when the teeth are being retarded by the resistant substance. The force is released as soon as the antagonizing surfaces come in contact; in fact, before they come in contact.

The amount of the force exerted by the muscles is dependent on the resistance offered, by muscle sense, and by voluntary control. While it is possible for normal muscles of mastication to exert 300 or 400 pounds pressure, this amount of force is used only when the character of the food requires it. The jaws do not work as a hydraulic press whose piston has as much force behind it in crushing an empty tin can as in crushing an iron casting. In the mastication of a meal of consommé, creamed sweetbreads, mashed potatoes, cottage cheese salad and ice cream, it is doubtful that a total of 400 pounds of energy is expended during the entire meal. This is the typical diet of the patient being treated for "traumatic occlusion."

In estimating the total amount of "overstrain" exerted three times a day on the teeth of the average patient, it is doubtful that more than a total of forty-

9. Mackenzie, W. C.: Action of Muscles, New York: Paul B. Hoeber, 1918, p. 4.

10. Footnote 9, p. 5.

11. MacMillan, H. W.: Clinical Application of Some Anatomical Facts to Dental and Oral Surgery, Bull. Chicago Dent. Soc., 10:22 (April) 1925.

five minutes out of the twenty-four hours is actually consumed in the preparation of food for deglutition. These forty-five minutes are divided into three periods of fifteen minutes each, separated by rest periods of from four to fourteen hours for recovery from the "traumatism" sustained. The "working" periods of fifteen minutes each are further broken into by "time out" intervals for conversation. Etiquette demands that the power of mastication be applied gently, without visible or audible manifestation. Special gnathodynamometers with micrometer gages should be used in obtaining records of the modern flapper on a diet. While this may appear exaggerated, normal stresses must be ascertained before the term "abnormal" can be used.

It has long been recognized clinical practice to disocclude teeth which have become too loose for compensatory abrasion. When the supporting structures have been destroyed to the extent of loosening the tooth, occlusal strains hasten the time of extraction. But this paper does not deal with the treatment of loose teeth. It is concerned only with "traumatic occlusion" as an etiologic factor. I assert that pyorrhea alveolaris cannot be produced on a "perfectly healthy tooth" by forces exerted by all the power which can be generated by well developed muscles of mastication in the preparation of the most resistant foods for deglutition.

"Normal vertical relationship to the line of occlusion."[12] This rule, tabulated as one of the "conditions found to be essential to a complete normality of occlusion," must have been formulated on the assumption that the teeth should receive the strains produced by mastication parallel to their long axes. Observations in comparative anatomy show that this is not true. The horizontally placed incisors and cuspids of the suina receive the forces of incising and tearing at right angles to their long axes. The combination of stresses in the anterior part of the mandible, caused by strains received at right angles to the horizontal incisors and by twisting unilateral strains produced by the powerful leverage of the mandibular cuspids, has produced thick heavy labial plates supported, in turn, by a heavily reinforced bony union of the two halves. In the proboscidea, the tusks are classified as incisors. In the act of uprooting trees and of carrying heavy mahogany logs, the force is received at right angles to the long axes. Pyorrhea alveolaris has not been observed in the incisors of the hog and elephant as the result of overstrain.

Primitive man with his edge-to-edge bite, employing cooking little or not at all, having no use for etiquette or salad forks, employed many muscles besides those attached to his mandible for incising. Using the orthal type of movement, the incisor teeth were forced into the resistant food, holding it firmly while a mouthful was wrenched off with the hands pulling at right angles to the long axes of the teeth. The triceps and the muscles attached to the occipital bone were just as much concerned in the operation as the masseter and internal pterygoid. The leverage exerted on the adolescent jaw, in the region of the ramus, by the forces employed in these various phases of incising and masticating supplied the stimuli for the "completer development" of Roux.[13] The stimulation afforded by the strains received at right angles to the long axes of the incisors of these primitive people produced

12. Footnote 3, p. 68.

13. Russell, E. S.: Form and Function, New York: E. P. Dutton & Company, 1917, p. 320.

the local resistance, resulting in almost complete absence of disease.

"Normal occlusal surfaces either natural or restored capable of coordinating perfectly with opposing surfaces."[12] Is it necessary for antagonizing teeth to be in contact or in "coordination" in order to function?

A feature of the dentition of the carnivora is the distance between the occlusal surfaces of the maxillary and mandibular bicuspids when the jaws are closed. Apparently, the careful coordinating of opposing occlusal surfaces was not one of the "selective" methods employed in the development of this most efficient dental machine. The distance between the antagonizing teeth shows that opposing teeth function even though they cannot be brought into contact. The high state of health of the gums of these animals in their native state shows that "occlusion minus" is not a specific cause for pyorrhea alveolaris.

In a 4-year-old steer, the mandibular incisor teeth may be worn to the gums, affording a beautiful example of wear with no opposing teeth. Examination of the teeth in the bicuspid region shows such degrees of wear that the antagonizing teeth may lack 2 or 3 mm. of meeting. The fact that wear continues in spite of the lack of occlusal contact shows that the teeth are still in use and continue to prepare the rougher foods for finer mastication by the molars. "Inharmonious wear of the occlusal surfaces" seems to be one of the distinguishing characteristics of a complete normality of occlusion in the pecora. In this connection, it is interesting to read that "traumatic occlusion" does not occur in the lower animals.[14]

14. Nokes, I. D.: Comparative Anatomy as Applied to the General Practice of Dentistry, Pacific Dent. Gaz., 25:468 (Aug.) 1927.

In the human being, inharmonious occlusal surfaces are among the prevailing characteristics of a denture which has been worn by abrasive food. At times, the maxillary and mandibular teeth of one side may have the cusps worn off entirely, while the cusps of the opposite side are only slightly abraded. The "overused" side, while being used, is invariably in the better state of health. Inharmonious wear prevails in the human being to such an extent that it must be considered as a normal rather than an abnormal characteristic.

If, for some reason, such as loss of a tooth or extensive caries, the chewing habits should be changed from the abraded side, the periodontium will degenerate on account of disuse atrophy. The resulting "pyorrhea" may be erroneously diagnosed as being caused by "traumatic occlusion" on account of the worn abraded tooth surfaces, when observation and inquiry into the history reveals that the wear was produced by the habits of years gone by. This diagnostic point should be emphasized. The mere presence of a bridge or crown on a tooth does not necessarily mean that the teeth are in overstrain. Checking over the chewing habits carefully may reveal that the patient never chews on that side. In the making of motion picture studies of the different phases of mastication, it has been most unusual to find a patient who chews with the same facility on either side. Ofttimes, an extraction, a capped pulp or some inefficient prosthetic contrivance may cause the patient to chew for years in a very restricted region. In estimating "abnormal stresses," these facts must be taken into consideration.

"Any tooth which is without mesiodistal support is one which will eventually suffer from the effects of traumatic

occlusion."[15] Are the teeth on either side of the diastema more prone "to degeneration of the investing tissues"? Did Nature bungle by not providing posterior support for the maxillary and mandibular third molars? According to this statement, the teeth should have been arranged in a circle, shoulder to shoulder, in conformance to that state motto, "United we stand, divided we fall." The single opposing molars of the proboscidea, the complete separation of the teeth of the carnivora and the numerous examples of healthy teeth in the human being which have no approximal contact are examples which do not seem to be in accordance with the empiricism quoted above.

The theory that each tooth functions independently of its neighbor; that the type of supporting osseous structure varies with the requirements of the individual tooth; that this osseous structure is sufficiently adaptive to alter its external contour and internal architecture in accordance with any change in the structure and function of the individual tooth; that the local resistance of the periodontium of this individual tooth is dependent on the stimulation furnished by that tooth and its antagonist, not by its approximal neighbors, and that the muscles which enable the tooth to function are under the direction and control, for the most part, of the same nervous mechanism that controls the caliber of the arteries, the processes of digestion and the phenomenon of respiration, has much more to support it in the realm of philosophy and in the field of experiment.

" . . . the hundreds of pounds pressure which the muscles of mastication can exert are distributed more or less evenly throughout the arches."[16] This statement, used as a part of the attempt to prove the "doctrine" of traumatic occlusion as an etiologic factor in the production of pyorrhea alveolaris, shows the danger of using an unproved theory as the major premise. The part of the quotation, "are distributed more or less evenly throughout the arches," is derived from the old theory of bilateral balance and three-point contact, which have been shown to occur neither in the lower animals nor in man during the act of forcing the teeth through tough resistant food.[17] During normal mastication, the food is not "distributed more or less evenly throughout the arches." The forces of mastication are distributed unilaterally, not bilaterally.

The statement that the human temporal and masseter muscles of *both sides* exert their power in the final biting and crushing movement is not anatomically correct, nor could it occur mechanically.[18] When chewing is performed on the right side, the excursive movement across the antagonizing surfaces is from right to left. In chewing on the left side, the movement is reversed. The muscles of mastication of one side are designed to move the mandible in a direction opposite to that produced by the muscles of the other side. One might as well claim that man could walk backwards and forwards at the same time.

In order to offer an explanation of the anatomic mechanisms involved in these new conceptions of mastication and to provide a rational explanation for the

15. Endelman, Julio: Special Dental Pathology, St. Louis: C. V. Mosby Company, 1927, p. 327.

16. Footnote 3, p. 65.

17. MacMillan, H. W.: Unilateral vs. Bilateral Balanced Occlusion, J.A.D.A., 17:1207 (July) 1930.

18. Clapp, G. W.: Mechanical Side of Anatomical Articulation, Dent. Digest, 15:813 (Dec.) 1909.

"balancing" phenomena which have been claimed in the past to be produced by "three-point contact" and "the opposite or balancing side," the term "unilateral balance" has been advanced and defined as the state of equilibrium produced by the muscles of mastication of the working side which enables each half of the lower jaw to function independently of the other half as far as any support from the teeth of the opposite side is concerned.[17]

The forces of mastication can be "distributed more or less evenly throughout the arches" only by voluntarily placing the teeth in "centric" occlusion and then exerting pressure. Centric occlusion plays no part in the functional excursion of the mandible. Centric occlusion cannot occur with food between the teeth. Centric occlusion does not arbitrarily mark the end of the functional excursion, as some individuals go beyond this "centric" point in their normal movements of chewing. In the average patient, during the mastication of hard, resistant cereal foods, the teeth on the working side are brought neither into occlusal contact nor as far as centric occlusion at the end of each individual masticatory stroke. In fact, the teeth make repeated attacks on the bolus and fall far short of reaching either occlusal contact or centric occlusion until after the food becomes softened and fluidized. Similarly, bilateral balance cannot possibly occur during the early stage of food preparation, which is the very time when the maximum amount of muscular effort is expended. The time when the "hundreds of pounds pressure" can be "distributed more or less evenly throughout the arches" is only during centric occlusion, which, at best, is only a descriptive term indicating the position of the mandibular teeth held, voluntarily, against the maxillary teeth in a median position with the muscles producing right and left rotation, protrusion and retrusion in equilibrium. This position is the antithesis of function.

Observations in comparative physiology indicate that the bolus is distributed over a small region, unilaterally. If the muscles are physically able to develop "hundreds of pounds pressure," this tremendous force is exerted unilaterally by the muscles of the working side on two or four antagonizing teeth, the periodontium of which is "built for emergencies at critical junctures."

## CONCLUSIONS

Inadvertent biting on a shot in eating game may produce a traumatic pericementitis. The presence of a filling or a crown in supra occlusion may produce a traumatic pericementitis. Teeth too loose for autocompensatory abrasion are traumatized in chewing. May these examples serve to emphasize that this paper is not concerned with the treatment of loose teeth or the etiology or correction of acute traumatic pericementitis? It is concerned only with the claim that "traumatic occlusion" is the predominating factor in the etiology of that symptom complex, pyorrhea alveolaris. To be more specific, it is my opinion that the tactile sense of the pericementum, the adaptive mechanism of the alveolar process to stress and strain and the somatic and autonomic nerve systems regulating the power furnished by the muscles of mastication are so perfectly coordinated that the "degenerative process" said to be initiated by "traumatic occlusion" could not be produced on "a perfectly healthy tooth" in the exercise of the basic and primitive function of mastication. It is my opinion, based on reason, experiment and clinical evidence, that teeth in malocclusion or teeth supporting bridges do

not develop pyorrhea alveolaris as a result of forces induced by the preparation of food for deglutition. I am equally certain that masticatory effort is unrelated to "tic douloureux," "sensitiveness to heat and cold," "formation of salivary calculus," "erosion," "approximal dental caries," "pulp infection," and "calcific pulp deposits."[5]

Since the introduction of the "doctrine" of "traumatic occlusion," there has been no diminution in the ravages of pyorrhea alveolaris. On the contrary, distinct harm is being done by directing the attention of the profession from the greatest corrective measure for alveolar border disturbances that lies in the field of the dentist—the stimulation of exercise in the prevention of disuse atrophy.

Pyorrhea alveolaris has not been produced experimentally by "traumatic occlusion."

In the published case reports on "traumatic occlusion," the dental roentgenograms, the gnathodynamometric readings and the patient's dental and general history have not been checked over by impartial observers.

The doctrine of "traumatic occlusion" is founded on theories which have been disproved.

The clinical practice of distoclusion has neither prevented nor cured pyorrhea alveolaris.

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DISCUSSION

*Paul R. Stillman, New York City:* There is always an element of greatness in the character of any man who is big enough to realize that he does not know. Thomas A. Edison has been quoted as having replied to a lady who exclaimed to him on the vastness of his knowledge: "Madam, the sum of human knowledge does not amount to a one millionth part of 1 per cent." The application of this thought to this paper is the fact that Dr. MacMillan begins the paper with the epigram: "Prejudice is a defense reaction of ignorance. You're apt to be 'down on' what you're not 'up on'." On my first reading of this paper, I began to feel like the small boy who hid behind the woodshed while his mother repeatedly shouted his name. The boy finally came out of his hiding place, and, when his mother scolded him for not answering promptly when he was called, replied: "I didn't know that you wanted me, Ma; I thought you were just hollering." Dr. MacMillan admits by the use of this epigram that he has "prejudice" regarding the thing he calls "traumatic occlusion." And for this I do not blame him. I would, myself, be decidedly against accepting the distorted picture of the thing as he has drawn it. For there could not be a more grotesque and prejudiced interpretation of my own beliefs regarding traumatic occlusion than has been presented here today. And whose fault is it that Dr. MacMillan has presented a caricature of this subject? That which Dr. MacMillan has presented here about traumatic occlusion has been derived from the writings of a very few men, and my own in particular. The fact that he has misunderstood, and has misinterpreted these writings, places the burden on the intellectual clarity of the writings, and not on the essayist. "You're apt to be 'down on' what you're not 'up on'." Whenever I have had to listen to some man speak, or to read some published article, and the words did not convey to my mind the thing he was attempting to make me comprehend, I concluded that the fault was not so much in my own lack of intelligence as in the inadequacy of his words to convey his thought. I have spent more than twenty years of close application to the study of the function of the teeth. In the very beginning of this study, there were no writings on this subject, except a scattered allusion occasionally found, and these never more than a paragraph in extent. In 1917, I published a paper under the title "Traumatic Occlusion," and it was several years later before any notice was taken of it by other writers. The conclusions of Dr. MacMillan in this paper regarding traumatic occlusion, as he has presented them here, would find us two not so very far apart in regard to our beliefs on this subject had this paper been read in 1917 instead of 1930. And I am willing to admit that I did not know very much about traumatic occlusion in 1917. Turning to Dr. MacMillan's "conclusions" as expressed in his

paper, we read: 1. "Inadvertently biting on a shot when eating game may produce a traumatic pericementitis." Here is a statement with which the whole world, dentists and laymen, will agree with Dr. MacMillan. Many have experienced it. Assuredly, it will traumatize the pericementum. But let me ask, what would happen were the stress, which is admittedly in excess of the normal, to be repeated successively many times a day over a period of several months? Would the acute traumatic pericementitis remain a traumatic pericementitis, or would chronic degenerative processes be set up to destroy the pericementum and the border of the alveolar bone; and would pus then flow? I think these things would happen and I think Dr. MacMillan would call the condition a chronic traumatic pericementitis then. Or, for want of a better term, would he call the condition pyorrhea alveolaris, as most of us would do? 2. "A filling, or crown in supra-occlusion may produce a traumatic pericementitis." In my experience, this pathologic expression of a reaction to injury is an inevitable sequence of such a tooth relationship, if trauma is long enough sustained. The very term supra-occlusion implies an abnormal stress, and it is quite unnecessary to make any attempt, as Dr. MacMillan implies should be done, to calculate how much in excess of normal this stress may be. 3. "Teeth too loose for compensatory abrasion are traumatized in chewing." Again, Dr. MacMillan has stated the obvious. But what he has failed to take into account is the series of pathologic changes which have led up to this state of mobility. What about the interval of time which intervened between the one condition, traumatic pericementitis, and the described condition "a tooth too loose for compensatory abrasion in chewing"? Why did not the compensatory abrasion take place before the tooth became loose and thereby forestall the loosening? This point Dr. MacMillan did not mention, and it is a very important one; and he may be under the impression that such compensatory abrasion takes place as a general rule. It is, as a matter of fact, rarely that we find true and complete compensatory abrasion. Teeth do not loosen spontaneously except for traumatic causes, or through the ingestion of a metallic poison, such as mercury, or by scurvy or some other severe systemic disease. Pulp disease may at times induce mobility. But especially and frequently teeth do become loosened through such an injury as supraclusion, and this is recognized by Dr. MacMillan in his paper. We call the supra-occlusion which brings this about "traumatic occlusion." But his term "supra-occlusion" carries the thought. What Dr. MacMillan evidently has failed to appreciate is the fact that many slight but nevertheless definite cases of supra-occlusion are to be found in the average natural dentition. These are not always caused by the presence of inlays and crowns, but are the result of abnormal occlusal form or slight malposition of teeth which have more or less correct form. A rotated cuspid or deviated molar cusp will produce essentially the same trauma as will the tooth in which an overcontoured inlay has been placed. This traumatic relationship is not always expressed in centric occlusion, the normal posture of the jaws when in rest, but will be found to be active in one or more of the excursive movements of the mandible. The fundamental effect is the same. 4. "That tactile sense of the pericementum, the adaptive mechanism of the alveolar process to stress and strain, and the somatic and autonomic nerve systems regulating the power furnished by the muscles of mastication are so perfectly coordinated that the degenerative process said to be initiated by traumatic occlusion could not be produced on a perfectly healthy tooth in the exercise of the basic and primitive function of mastication." If Dr. MacMillan were familiar with periodontia, he would know that neither I nor others who are active in this work claim that the periodontium of a "perfectly healthy tooth" will show degenerative changes under the influence of traumatic occlusion, nor will a perfectly healthy tooth show any pathologic change. I do believe that traumatic occlusion will eventually produce some form of damage, either in the tooth itself, or in the nerves and vessels supplying it, regardless of the state of health of the periodontium. If he wishes to regard the as yet unidentified aberrations which, together with traumatic occlusion, lead to periodontal disease as being more important than the traumatic occlusion, I will not quarrel with him. I nevertheless maintain that these same aberrations do not lead to periodontal disease when the occlusion is mechanically physiologic, and it is on this basis that I predicate the fundamental relationship of this factor to these disease processes of the periodontium. But let us revert to Dr. MacMillan's state-

ment that the tactile sense of the periodontium provides an automatic safeguard against self injury during the occlusal contacts and excursions of the mandible. Please notice that I omit mention here of the word "mastication." The injuries which are produced by traumatic occlusion rarely occur while food is between the teeth, but they do occur when the teeth have met each other after crushing through a morsel of food, or when they have met without food having been between them at all. In the natural dentition, if there is gross or even slight malposition or malformation, traumatic occlusion develops gradually as the teeth come into full eruption, and is therefore not perceptible to the nerves of the periodontium, as in the case when a sudden supra-occlusion is introduced, for instance, by means of an inlay. There is, therefore, no adaptive avoidance of biting on that particular spot which is producing the traumatic occlusion, as Dr. MacMillan assumes would be the case. I am beginning to see that there is a miscomprehension of traumatic occlusion which extends far beyond the mind of Dr. MacMillan, and is more or less general among many in the profession. Occlusion means the act of bringing the teeth together with more or less muscular force; as, for instance, in the act of swallowing. Some observers doubt that the teeth are brought forcefully together, tooth on tooth, during mastication. Dr. MacMillan is one of them. It may be so, but we are not concerned with that proposition here. My own opinion is that teeth, even those having a pronounced incoordination in their occlusal relations, do little if any injury to themselves in the act of mastication. It is here that the defensive tactile sense of the teeth comes in; for not only do teeth have tactile sense against foreign bodies, such as gun-shot in the food, when its presence is known, but they also employ this same faculty against injury of tooth on tooth. But as Dr. MacMillan says, mastication is a "basic and primitive" function, and I add that it can never be suspended so long as any occluding teeth exist in the mouth. The fact is that human beings chew their teeth very much more than they chew their food. And it is this almost universal act of exerting pressure or abnormal stress (I persist in using this term), tooth upon tooth repeatedly, times and times without number daily, which produces injury, and this injury I have chosen to call traumatic occlusion. There are varied reactions to physical injury. Some are immediate and at the site; some are far removed. No need of examples or analogies here. Dr. MacMillan's medical degree should guarantee his knowledge of this; whether he is certain, as he states, that in the "masticatory effort" (and here I interpret he means closing the teeth with muscle force applied), the act of closing is etiologically unrelated to such clinical expressions as "tic douloureux," "sensitiveness to heat and cold" (dental pulp), "formation of salivary calculus," "erosion," "approximal dental caries" and "calcific pulp deposits," I care not a whit. I am of the opposite opinion, and I believe that "'tis so" and "'taint so" will not get us anywhere in this discussion. It is obvious from Dr. MacMillan's statements that he has no knowledge of modern treatment in periodontia, and he states some very misleading half truths, as, for instance: "Since the introduction of the 'traumatic occlusion doctrine' there has been no diminution in the ravages of pyorrhea. On the contrary, distinct harm is being done by directing the attention of the profession from the greatest corrective measure for alveolar border disturbances that lies in the field of the dentist—the stimulation of exercise in the prevention of disuse atrophy." Now let us analyze this. In the early part of his paper, Dr. MacMillan pokes his finger at me a bit in taking exception to my use of the term "abnormal stress." He states that I used the term "with as much assurance as though the actual number of pounds pressure of overstrain had been definitely determined." That is no argument coming from a physiologist. It might more properly be used by a mechanical engineer who is supposed to know a little of physiology. But let it pass. Again he states: "Since the introduction of the 'traumatic occlusion doctrine,' there has been no diminution in the ravages of pyorrhea alveolaris." In this instance, he speaks *ex cathedra*. He pontificates. The facts are that he is in error. In other words, he is misinformed. My own observations have led me to form an opinion diametrically opposite to that. Never, until long after the introduction of the traumatic occlusion hypothesis, had enduring high health been possible through effort directed at the suppression of periodontal disease. Never, until treatment based upon the theory that a perfectly coordinating occlusion liberated health impulses and induced healing had there been successful treatment of pyorrhea alveolaris. And by successful I mean that

kind of health which is self perpetuating through normal functional exercise. I see such results from my own efforts. I see them in the college clinic. I see them from the hands of colleagues. To this extent, there has been a "dimunition in the ravages of pyorrhea alveolaris." To quote Dr. MacMillan again: "On the contrary distinct harm is being done by directing the attention of the profession from the greatest corrective measure for alveolar border disturbances that lies in the field of the dentist—the stimulation of exercise in the prevention of disuse atrophy." If the attention of the profession is being directed by a discussion of traumatic occlusion from "the greatest corrective measure," Dr. MacMillan seems to me to be in duty bound to tell us something about the nature of this "greatest corrective measure." Is this greatest measure a new cure? Traumatic occlusion is a disturbed functional relation of the teeth which produces harm to the tissues, and its correction provides the means for natural stimulation through normal exercise of the teeth. Fully rounded exercise of the part, any part of the body, will preclude the development of atrophy. And only when we provide normal function through the correction of such an etiologic factor as is traumatic occlusion can we expect free functional use to produce true health and structural regeneration and development. I quote again: "The doctrine of traumatic occlusion has been founded on theories which have been disproved." What are these theories? Dr. MacMillan does not tell us, nor does he tell us who has proved them to be false. Does he refer to his present essay? The only words against traumatic occlusion with which I have acquaintance are nothing more than dogmatic statements, expressing a personal opinion, and remain without evidence to back them up. I wish to quote the following as being a concise statement of my own theories and practice. It comes from one high in authority: In his address of welcome to the members of the Clinical Congress of The American College of Surgeons, held in Chicago, Herman L. Kretschmer, M.D., F.A.C.S., was quoted as follows: "We all agree that effective and rational treatment must be based upon our knowledge of the structure and mechanism of the human body. We can help the sick and maimed in only one way—by aiding and abetting the natural defensive and recuperative mechanisms of the human body. Our methods must be based on a knowledge of these mechanisms." And here I should add that the correction of traumatic occlusion as it is understood at this time provides a compensating functional relation for the teeth, and makes possible that physical freedom which is found only in a healthy body. Dr. MacMillan made much of my failure, and that of other writers on traumatic occlusion, to measure the degree of overstress; he demands that we speak in terms of pounds pressure. Quoting again: "Since the maximum amount of stress that the periodontum is able to withstand without injury has never been ascertained," he asks, "how can the term 'abnormal' be used in the definition which should fix or prescribe clearly and authoritatively?" In my opinion, this is the only valid argument in his paper. Well, let us take a little excursion into Dr. MacMillan's own chosen field of medicine. In a recent article on rheumatic disease[19] by Philip S. Hench, M.D., of the Mayo Clinic, we find the following statements, which seem decidedly pertinent: Dr. Hench, writing under the heading "Chronic Traumatic Arthritis," states: "Chronic trauma that can produce arthritis may be from two sources; exogenous, from the outside of the body, such as occupational trauma; or endogenous, from within the body, such as from postural defects, curvature of the spine, flat feet, and particularly obesity. It is probably rightly said that every occupation has its special trauma and this is amply illustrated by a variety of types of traumatic arthritis: 'farmer's back,' 'golfer's shoulder,' 'nun's knees,' 'miner's elbow,' 'baseball fingers,' and even 'card player's wrist.' Assuredly infection plays a minor part in these cases. The most common form of endogenous chronic trauma is that from obesity. *The normal body architecture, particularly of the lower portion of the spinal column and legs, has a reserve capacity to uphold a certain amount of excess weight for a certain number of years. The period of compensation may vary and the introduction of other added insults may induce decompensation.* Obesity, well-borne and painless at the age of thirty, may be productive of many an aching joint at the age of forty-five, when the stage of early senescence of tissue is approached. Of

19. Hench, P. S.: Treatment of Dental Foci in Chronic Rheumatic Disease, Dent. Survey, June, 1930, p. 19.

course, true chronic traumatic arthritis from obesity must manifest itself only in the weight-bearing joints: the lumbar spine and sacro-iliac joints, knees and feet. The hips seem to escape, probably because of special protective anatomic development. In general, in chronic traumatic arthritis, dental foci are treated as an unrelated problem, conservatively or radically, as oral conditions alone seem to warrant. *However, because infection seems prone to localize as a secondary implantation in sites of chronic trauma,* due care must be given to eliminate frank infection." (Italics mine.) The analogy of chronic traumatic arthritis to chronic periodontal disease is not new, and in this instance it is so strikingly stated by Dr. Hench that comment is superfluous. However, it is desirable, in view of Dr. MacMillan's statements just referred to, that we call attention to some things not directly mentioned. For instance, Dr. Hench is not only a physician but also a member of the staff of an institution famed for its research work. *He* does not find it necessary to measure the number of "pounds pressure" which the knees and ankles of his patients will tolerate without injury. Dr. Hench frankly states that there is a *variation* in these factors, but he does not hesitate to ascribe to overstress alone, of an undefined amount, the introduction of a definite disease process. Nor do I hesitate to do the same thing as regards the teeth. Another statement in my quotation has a significance which it may be well to emphasize. Dr. Hench states that "infection seems prone to localize as a *secondary* implantation in the sites of chronic trauma." This is exactly what we see in the type of periodontal disease, designated by Box as chronic periodontitis complex. First, there are the noninfectious changes directly produced by traumatic occlusion; following these, there occurs, in the course of time, as a result of the continued irritation, a chronic suppurative infection, call it pyorrhea or periodontoclasia, whichever name suits you best. With this last analogy, I close my defense in "The Case Against Traumatic Occlusion," and I rest my case here.

*F. A. Bricker, Hollywood, Calif.:* I do not think the periodontist has ever spoken of producing periodontoclasia from overstress in normal function or in the act of masticating food. As I understood the situation, most of the trauma was produced while the mouth was empty, from teeth that did not get rootwise stress while the mouth was empty; in other words, that trauma did not occur except when the mouth was empty. I do not know whether Dr. MacMillan did not bring that out clearly enough or whether I have not recognized it. Nature will compensate for any stress, if it is normal stress. We say it is abnormal lateral stress produced under abnormal conditions. Therefore, we believe where bridge work is placed and there is overstress on abutment teeth, we do not get periodontoclasia under those conditions. We get an overcondensation at the apex or surrounding those teeth, from overstress, perhaps, but it is a normal condition.

*H. J. Leonard, New York City:* I cannot adequately express the admiration I have for this valuable work which Dr. MacMillan has done on the physiology of mastication. However, it seems to me in regard to the case of traumatic occlusion that he has not entirely hit the point. Traumatic occlusion, as I see it, is usually not a developmental condition in that it occurs to teeth as they erupt in the mouth. Traumatic occlusion develops from a shifting stress, from the fact that certain teeth are loose and other teeth are shifting in their positions, or that the teeth have become worn in such ways that they develop lateral stresses that they did not at first receive. Dr. Jensen, in his book on bone formations, makes a very interesting point regarding the inability of bone to resist stress which comes in a direction different from the stress for which it was developed. He speaks of the tremendous pressure stresses which the vertebrae sometimes receive, at times several hundred pounds from above downward. If a pressure stress develops from the side, such as develops in an aortic aneurysm, a pressure of perhaps not an eighteenth or a twentieth of the amount which that bone normally receives or can receive from above, it is very rapidly absorbed. The stresses we get in the alveolar process which develop traumatic occlusion occur during the time that the teeth are shifting in their positions. The shifting of the teeth is developing faster than these bone formations can occur.

*Arthur H. Merritt, New York City:* Dr. MacMillan emphasizes the question of the etiology of traumatic occlusion in connection with diseases of the periodontium. We see cases in traumatic occlusion as does Dr. MacMillan. The question he is raising is whether traumatic occlusion is productive of disease

in the periodontium, or is it, in most instances, a symptom of disease. From my own experience, extending over more years than I am sometimes willing to admit, I have come to the conclusion, over and over again, as I study the individual case, that trauma is the result of disease and not the cause of it.

*Dr. Bricker:* I will correct my remarks. I think that traumatic occlusion is the result of disease. You would not have traumatic occlusion if you did not have lowered resistance in the area to start with.

*Dr. MacMillan (closing):* The defense of Dr. Stillman merits more than passing consideration. Each statement should be taken up and analyzed carefully as too many men in the profession accept without reservation the statements of those who have distinguished themselves by originating new theories and practices. I am sure that Dr. Stillman will admit that he is as prejudiced in favor of traumatic occlusion as being the "predominating factor" in the production of pyorrhea alveolaris as I am prejudiced against it. He says that he did not know much about traumatic occlusion in 1917. May the second edition of his book make its hasty appearance. If a patient should inadvertently bite upon a shot "repeatedly, successively, many times a day," he would promptly develop an acute traumatic pericementitis and lose the tooth from pulp disturbance instead of chronic traumatic pericementitis or pyorrhea long before "a period of several months" had elapsed. How the patient could inadvertently persist in doing this over a period of months, I am not aware. The series of "pathologic changes which lead up to mobility" are subjects of other published papers which may be found under my name in the "Index of Dental Periodical Literature." As one of the "unidentified aberrations," disuse atrophy, that greatest local predisposing cause of disease even where "the occlusion is mechanically physiologic," is suggested. "The perfectly healthy tooth" quotation is from McCall. The empiric statements about the injuries produced by traumatic occlusion when there is no food between the teeth have not been qualified by experimental methods or logic. "The position of repose is neither complete closure nor opening of the jaws." The fantasy concerning excessive pressure of the teeth during swallowing is another hypothesis which should be given the dignity of a theory before it is used as proof. Why should we not measure the normal swallowing pressure? Then, if the sufferer exerts an "abnormal force," instruct him to swallow less vigorously. Dr. Stillman's assertion that I "stated some misleading half truths" in my paper is in itself a serious charge. From these personally supervised restricted clinics where the "ravages" have been checked, to my knowledge, there are no published reports of control patients who have received no form of treatment for pyorrhea other than grinding the teeth out of occlusion. The use of toothbrushes, dentifrices, mouth washes, scaling and all other forms of surgical treatment, massage, dietary corrections, and treatment of such general disturbances as may have oral manifestation must be eliminated to determine the true value of the grinding. If traumatic occlusion comes so close to being a specific cause of pyorrhea—it has been claimed that it is "the predominating etiologic factor"—why does not disocclusion alone effect a cure by removing the cause? Pyorrhea is becoming more prevalent not because people chew too much but because they chew too little. And now the climax of the argument for traumatic occlusion: The extensive quotation from Dr. Hench should be included in dental literature, first, because it is a beautiful example of how a scientific article should be written—clear, concise, specific, intelligible, logical; and secondly, because it emphasizes the effect of *continuous* pressure. Obesity produces a continuous pressure from which the tissues never secure a rest period. Walking, running, sitting, standing, sleeping, the weight is always there to a greater or less degree. Continuous pressure such as is present in obesity and is produced by benign tumors, aneurysms and tightly laced corsets, and which arises from employment of dental regulating appliances, produces resorption. The most ardent believer in traumatic occlusion has not this conception of the force which it is claimed is exerted upon the periodontium. "According to the views of certain authors, Jores in particular, the important thing is the intermittent character of the pressure. A continuous pressure favors resorption. But if this pressure is applied intermittently at long enough intervals, then, during the intervals without pressure, there is proliferation of bone in the exact place where

the pressure exerted its effect."[20] The rest periods between masticatory effort have been emphasized in the paper. If the case "for" traumatic occlusion rests upon the phenomenon of continuous pressure, it has a very poor foundation indeed.

20. Leriche, R., and Policard, A.: Normal and Pathological Physiology of Bone, St. Louis: C. V. Mosby Company, 1928, p. 100.

# THE CLINICAL SIGNIFICANCE OF TRAUMATIC OCCLUSION*

By CLAYTON H. GRACEY, D.D.S., Detroit, Mich.

So much of the clinical aspect of traumatic occlusion has been written into our literature from the experiences of numerous teachers, research workers and students that a paper covering the subject must be but an evaluation of their findings. With the exception of the pulpless tooth this phase of dental pathology has probably, within the last few years, been given more study than any other. I wish to give full credit to and express my appreciation for the painstaking efforts of men such as Stillman, McCall, Merritt, Box, Bricker and James, and to many others who have given to us so unselfishly the result of their experiences.

The preponderance of case histories, clinical and roentgenographic findings and voluminous writings of these men of unquestioned authority make it imperative that we, the profession of dentistry, recognize the importance and far reaching significance of that condition universally termed "traumatic occlusion."

Presenting a paper on traumatic occlusion signifies the acceptance of the term as descriptive and scientific. In our literature but few exceptions can be found to the use of the phrase. Hermann Prinz argues that the terminology is a mistake, being a contradiction within itself. To quote: "The cause is occlusion and the effect is trauma, consequently, occlusal traumatism, and not traumatic occlusion, should be used." This technical objection is not accepted generally, and most teachers and writers use the expression "traumatic occlusion" in dealing with this condition.

It is a fact, also, that a minority of clinicians and writers frown on focusing so much attention on this factor as a primary cause of disease of dental tissues. The ultraconservative, who are pleased to accept Nature's handiwork, consider it unprofessional to revamp or change in any way natural tooth form or the interrelationship of teeth. Some of the same objectors will oftentimes countenance restorations which are not in accord with the natural tooth form and which cannot be considered compatible with the demands of normal function. Especially is this so in the exaggeration of cusps and sulci or in their entire absence.

The acceptance of A. Leroy Johnson's axiom that "the law of occlusion is the basic principle of dentistry, the fundamental truth upon which the science is

*Read before the Section on Oral Pathology at the Midwinter Clinic of the Chicago Dental Society, Jan. 13, 1930.

built," brings the study of occlusion and its deviations into the field of every dentist.

In his report on five years' work in mouth hygiene in Bridgeport, Conn., Alfred C. Fones states that 98 per cent of school children examined within that period showed malocclusion. Conservatively, 95 per cent of these dentitions are constantly defying the basic principle of dentistry and its fundamental laws. What a potential influence such conditions are for the development of traumatic occlusion!

Very recently in the history of dentistry, in fact but a few years ago, the periodontist presented himself for recognition by the profession. With his acceptance came a keener and much finer perception of traumatic occlusion and a broader concept of its sphere in dental pathology. Almost from the beginning, he recognized the pathogenic possibility of this influence and accepted the fact that the source of periodontal and intradontal disease could very frequently be attributed to traumatic occlusion (intradontal, i.e., within the pulp chamber).

As an etiologic factor, traumatic occlusion has been more or less definitely established in the pathology of three distinct groups of tissues: (1) in the periodontium, including the gingival tissues and mucosa, the pericemental membrane and alveolar bone; (2) in those tissues within the pulp chamber which nourish the tooth, "intradontal" tissues, and (3) in the hard tooth tissues, the enamel, dentin and cementum.

Box defines normal occlusion as physiologic and as that condition in which the systems of forces acting on the tooth during occlusion are in a state of equilibrium, and do not and cannot change the normal relationship existing between the tooth and its supporting structure. This definition, though broad, does not recognize the possibility that pathologic processes in the blood, nerve and lymphatic tissues chambered within the tooth, or in the hard tissues of the tooth structure itself, might have their initiation through the force of an occlusion *not* in a state of equilibrium. This limitation of definition might be slightly misleading to the student, but was not so intended by Box. He is one of the most ardent teachers of the multiple types of pathologic conditions arising in dental tissues from traumatic occlusion.

Let us consider these areas of disease in the inverse order of their importance to clinical findings and study.

*Significance of Traumatic Occlusion in the Development of Dental Caries.*—Students of this subject offer for our consideration the definite statement that dental decay of certain types has its etiology in traumatic occlusion. Paul R. Stillman, in a very recent paper, states: "In these cases, the effect on the blood stream in the pericementum appears again to involve the pulp circulation, resulting in a deficiency which manifests itself in an increased susceptibility to certain forms of caries, as, for instance, cervical caries, both buccal and labial with a coincidental disturbance of circulation in the marginal gingiva, next adjacent."

In presenting illustrations of marginal gingival recession, Box indicates his conviction when he states in the accompanying descriptive text that "labial, cervical caries always occurs in the presence of a marginal gingivitis."

The coincidence of dental caries and gingival disturbances, particularly in certain areas is, as far as can be found, the only authoritative basis, to date, for accepting the premise that traumatic occlusion is a cause of dental caries. Innumerable clinical case histories re-

cording the presence of caries in this environment must convince us of a definite relationship. The positive significance of traumatic occlusion in the development of caries, from a systemic standpoint, is still problematic.

There can be no doubt about incipient caries having its influence in local conditions which have been created by traumatic occlusion. The utter lack of hygiene which prevails at the cervical and most crucial point in certain types of periodontoclasia, the excretion of fluids and deposition of acid-promoting mucin plaques and decomposed carbohydrates all combine for the dissolution of the tooth tissues. Just what bearing internal influences have on the development of caries under these conditions is, to date, most uncertain also.

*Significance of Traumatic Occlusion in the Etiology of Dental Pulp Involvements.*—The findings of our research workers in histology have definitely proved the intercommunication of the blood, nerve and lymphatic supply of the periodontal tissues with the pulp tissues; hence, a functional disturbance of either group reacts on the other, usually with quite positive symptoms in both regions. Occlusal stresses producing congestion of the periodontal tissues must be either directly or indirectly developing a like condition in the intradontal tissues.

How often in those teeth which carry large metal fillings do we find pulpitis, with hypersensitiveness and other neurotic symptoms, relieved on the adjustment of excessive occlusal stress. This does not necessarily signify that the filling, on its insertion, had been so built as to be in traumatic occlusion. A natural tooth under mild plus occlusion, presenting negative symptoms of periodontoclasia, on being burdened with a metal restoration, with its attendant shock of operation and additional thermal conductivity, but with no appreciable occlusal accentuation, will readily develop a pulpitis of acute activity with all the symptoms of a typical traumatic occlusion. The result has been a localized congestion in the apical region of such a tooth, causing a more extreme malocclusion of the tooth with its antagonist.

We must not overlook the fact that excessive longitudinal and oblique occlusal forces are carrying their tremendous impacts through the root end and directly compressing and impinging with each blow, nerve and blood tissues in exit from the dental foramen. It is not hard to conceive of Nature's inability to protect these tissues from a force which knows no limit up to the maximum degree of mandibular muscle stress. Her efforts in this behalf are sometimes indicated by the diffused and morbid depositions of calcium salts frequently found in the cancellous bone about the apices of teeth so abused and appearing as radiopaque blotches in the roentgenogram.

But the result of such trauma is more often evidenced in that confusing periapical osteoclasia so frequently interpreted through the roentgenogram as an incipient granuloma. This abnormality is frequently found involving the bone at the apices of teeth having otherwise an unusually strong bone support and presenting negative clinical symptoms of overburdening in the gingival region. Extreme sensitiveness and tenderness are coincident clinical symptoms.

We have all experienced discomfiture on opening a pulp chamber for the treatment of a nerve with roentgenographic and clinical symptoms of complete putrescency, or necrosis, only to find vital sensitiveness. A further diagnosis invariably proves the presence of pulpitis and a disturbance of the apical vascular

system, due to an excessive occlusion. This has caused an acute absorption of the alveolar periapical bone. With little or no infection to overcome, with the area circumscribed by normal bone, regeneration at this point, after treatment, is accomplished with less effort and in a shorter period than in the seat of other alveolar bone absorption.

Traumatic occlusion again frequently expresses itself through the development of hard deposits in the pulp chamber, sometimes diagnosed through the roentgenogram as calcific degeneration or the presence of pulp stones. These foreign bodies are tolerated for years in some teeth without evidence of discomfort. Their growth will, at times, advance faster than the recession of the pulp, and extreme pain ensues from the pressure. Because of high resistance and the general good character of the tissues, these pains are often not localized, evidencing themselves as reflexes in the nerve trunk supplying this region. In most instances in which a patient has an obscure pain or neuralgic tendency in the dental organ and no caries or pulpless teeth in the area involved, it is good practice to assume that there is some occlusal disturbance directly affecting the nerve tissue or indirectly affecting it through congestion or irritation, as shown by calcific deposits.

Periodontists have often noted the change in the shade of natural teeth the supporting tissues of which are under treatment. This change is particularly noticeable in teeth which have as neighbors porcelain teeth with their fixed color. A great deal of this variation, of course, is accomplished by intensive polishing, removal of deep-seated stains and change in the density and color of the neighboring gingival tissues, yet it is within reason to attribute some of it to the coincident recession of inflammatory conditions within the tissues of the pulp chamber.

The translucency of dentin and enamel, being appreciably affected by the density and depth of color of the tissues within the pulp chamber, a congestion herein induced by traumatic occlusion or other factors, will deepen or dull the natural shade and quite noticeably affect the color.

*Significance of Traumatic Occlusion in Diseases of the Periodontium.*—This relationship is so evident and so in harmony with clinically proven facts that a study of the significance of traumatic occlusion brings us at once to a consideration of the symptoms of periodontoclasia. These symptoms are readily noted by changes in the character of gingival tissues, as to both color and density, pocket extensions, alveolar bone destructions, the presence of pus and tooth mobility. Box, in his article entitled, "The Twenty Signs of Incipient Periodontal Diseases," presents to us clearly the clinical and roentgenographic evidence of functional disturbances in these various tissues. It is hard to reconcile his use of the word, "incipient," in the title phrase, because most of the conditions illustrated herewith are advanced lesions.

These signs can be conveniently arranged for study into five divisions:

1. The first, which might be termed the visible, carry tissue changes at the gingiva through the range of simple congestion, recession, the development of so-called traumatic crescents, the Stillman clefts, festoons, linear depressions in the alveolar mucosa, and the surface changes in the mucosa from a stippled to a glazed epithelium, also epithelium nodules found very infrequently and located several millimeters from the gingival line on the mucosa tissue.

2. The roentgenographic signs are the changes in the alveolar bone as indicated by the radiolucency of the structure, the widening of the pericemental space, the density or thickening of the lamina dura and the absorption of the alveolar crest.

3. Both tactile and visible signs include tooth mobility of two degrees, classified as No. 1 and No. 2.

4. Tissue detachment, presenting its evidence through pocket formation, is in the fourth division.

5. Lastly, the microscopic signs are considered (the presence of pus in the secretions in these pockets).

After presenting all these symptoms or signs of periodontal lesions, in a paper on traumatic occlusion, it is well to check what might be advisedly construed as an overenthusiasm or seeming partiality for but one of the many known etiologic factors in the development of such pathologic conditions. We must sound at this time a precautionary note against focusing too much attention on just one manifestation that may probably be, and often is, but a sequel of a deeper, more complex and remote cause.

The frequency of the coincidence of traumatic occlusion in the presence of each and all of these symptoms is a valid excuse for suggesting their consideration in this article. It is the experience of most periodontists and also many men in general practice, who are conscientiously determining the etiology of periodontal lesions, that traumatic occlusion evidences itself consistently in one or several of the signs above enumerated. It is also their experience that, on the relief of a clinically proven excessive occlusion, these symptoms, in both the soft and the hard tissues, are very frequently improved, oftentimes to the extent of disappearing entirely without the concomitant influence of other remedial operations.

The most ardent disciples of the complex of traumatic occlusion do not ask us to take their word for its significance in dental pathology. It is repeatedly suggested, where certain periodontal lesions exist which might provide a suspicion of excess occlusion, that an experiment be made. We are urged not to change the gingival skyline by surgical procedure, instrumentation, artificial stimulation, change in diet or mastication, until the involved teeth have been brought into a state of physiologic rest or even taken out of occlusion by grinding. This, of course, should be carried out judiciously and with due respect for certain well known principles of occlusal adjustment.

Assuming that the teeth under experiment have been relieved of excessive stress, but other factors are unchanged, is it reasonable to expect an immediate improvement in the tone of the gingival tissues? The burden of proof as to the significance of traumatic occlusion to periodontal lesions would seem to rest squarely on the biologic response to such a test. In most instances, an improvement will be noted. Congestion probably associated with clefts or breaks in the normal gingival curve will be improved, the cleft often disappearing entirely and the normal gingival curve being reestablished. But Nature's best effort in such an experiment is often thwarted by the presence of deposits, accretions and other handicaps, too burdensome to overcome.

Another very interesting fact, also recognized in such an experiment, will be the improvement in the mobility of teeth, with a tendency to an elongation into plus occlusion again. Even when entire occlusion relief is afforded a second and a third time, there may later be an extension of the tooth into occlusion. This is due to the revived activity of the oblique fibers in the periodontal mem-

brane, which, under constant intermittent irritation, have become stretched, flabby and functionless. With the irritation removed, a natural tensioning of these bundles of fibers begins, and, owing to the angle of their attachment, from the surface of the cementum to the lamina dura, their pull on the tooth is outward and toward its antagonist. This continues until they are in physiologic tension and normal function, with a resultant tightening of the teeth in their alveolar bone support. If the mobility of teeth as a symptom of periodontoclasia is admitted, the significance of traumatic occlusion to this form of dental disease is again attested.

It must be conceded that traumatic occlusion in itself, uninfluenced by other agencies, could not be responsible for these deep-seated periodontal lesions, but a long continued intermittent irritation, through interference with the circulatory system in the structures involved, so lowers their resistance that the way is prepared for the invasion of ever present micro-organisms. Though seemingly paradoxical, this same intermittent irritation has also prepared the tissues for a sharp biologic response to treatment.

Excessive occlusion, as has been pointed out in numerous writings, is, in its primary stages, a stimulant and continues as one for a varying period of time. When stimulation develops into irritation is dependent on the loss of equilibrium between the mechanical forces at work and the biogenic tone of the tissues. Having for an extended period been subjected to this initial stimulating influence, the regenerative forces are developed into a hyperactivity, in consequence of which, when the irritant is removed through occlusal adjustment, the reaction is more definite and acute than though this preliminary overfunction had not existed. A differential comparison with the response to treatment of lesions having their initiation in nonocclusion might help to an appreciation of the significance of this reaction. In the latter condition, there is an entire absence of function so necessary for maintaining the health, plus a resultant lowered degree of biologic activity and regenerative potentiality. The influence of treatment is thus retarded and made more uncertain, which necessitates the institution of the most intensive artificial stimulation to compensate for the diminished recuperative powers.

If we acknowledge its presence in abnormal dental conditions, what are the influences acting on and the immediate significance of traumatic occlusion to the pathologic complex of the tissues of the oral cavity?

Anthropologists, through the study of tooth abrasion, wear and supporting bone changes, have given us proof that this has been a variable quantity throughout different periods of civilization. This influence is determined by the relationship between the incidence of traumatic occlusion in dental conditions and its toleration by the dental tissues.

The following conditions are very considerably influencing the incidence or prevalence of traumatic occlusion:

1. The predominance of congenital malalinement of dental arches and teeth.
2. The early loss of teeth, especially the permanent molars, with resultant tipping and drifting.
3. The use of hard golds and porcelain, which do not wear or flow under occlusal stress in dental restorations.
4. The placing of fixed bridges and crowns with highly carved and accentuated occlusal surfaces, incompatible with normal function.
5. The use of cast clasps and other retaining devices which are serving in the

capacity of orthodontic appliances and moving teeth out of position.

6. The complacency of the dental profession in the presence of incipient periodontal lesions that might often be attributed to traumatic occlusion.

The toleration of traumatic occlusion by the dental tissues, on the other hand, is influenced by:

1. An ever-deteriorating grade of tissue-building foods in use today.

2. An ever-lessening need for normal mastication with its attendant lack of functional activity.

3. A generalized lowered resistance induced by sedentary habits and an acceleration of activities.

However discouraging such introspection might be, there are many influences, both natural and man-made, which are active in counteracting the effects and forcing the subjugation of this menace to the health of dental tissues.

Bunting says:

> Of all affections there are none which may be so definitely and successfully prevented as those which affect the periodontal tissue. The hope of controlling periodontal disease does not lie in the treatment of advanced cases, but in the early recognition of incipient forms of the disease, which readily respond to preventive measures. Success in treating and controlling these conditions is dependent on the recognition of the most predominating factors in each case, and the institution of measures which will either remove the determining causes or reduce them to a state of inefficiency.

In this last sentence lies the justification of a hopeful outlook for the control of traumatic occlusion and other etiologic factors in periodontal diseases.

This responsibility cannot be delegated to that small group of dentists known as periodontists; it must be accepted by the prosthodontists, the crown and bridge and porcelain specialists, and by every member of the dental profession who is conscientiously building into his work that ultimate in health—normal function.

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# A SURVEY OF SYSTEMIC DISTURBANCES TRACEABLE TO INFECTED TEETH*

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BECAUSE medicine is a dynamic science, our ideas of the nature and the causative factors in many diseases are constantly changing. In the past twenty years, the possible relation of focal infection to almost every disease has at least been considered. Different observers hold widely different views concerning the rôle of foci of infection in the causation of systemic disease. This survey is an attempt to summarize the most competent medical opinions on the subject. Such an accounting at intervals is most important in the study of dental focal infection.

Dental infection may cause systemic disease by direct extension into adjacent structures or by metastasis to distant parts of the body. Infection may spread

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from a tooth into the alveolar process, producing a diffuse osteomyelitis; into the soft tissues, producing a cellulitis, or into the nearby lymph glands, producing a lymphadenitis. The maxillary sinus is not infrequently infected from the upper molars or bicuspids. Occasionally, a sinus thrombosis results from the direct extension upward of an acute dental infection. Rarely, the bacteria entering the blood may multiply and produce a true septicemia. No discussion concerning the rôle of the dental focus in such conditions is needed. The relation is too apparent.

Metastatic systemic disease is usually due to the actual transference of bacteria from the focus to different parts of the body. The organisms are carried by the blood or lymph stream to the point of localization, where they multiply and incite an infection of the tissues in which they have lodged. At times, the bacteria remain encapsulated at the focus and give off toxins which may produce functional or structural changes in different organs of the body. Systemic disease may also be due to the development of allergy from a focus of infection. Here, the individual becomes sensitized to toxins absorbed from the focus, so that further absorption of poisonous products produces the characteristic symptoms of the hypersensitive state. The discussion and difference of opinion about the importance of dental infection is concerned only with systemic disease of metastatic origin. Diseases of different organs of the body will be considered in detail.

### ARTHRITIS

Arthritis is one systemic condition which well illustrates the flux of medical opinion concerning the relation of dental infection to distant disease. It is one of the most common clinical conditions met with in medical practice. It is likewise the lesion most commonly seen in animals after the injection of bacteria from foci of infection.

We have gone through a period in which it has been thought that most cases of arthritis were infectious in origin. A few exceptions, such as gout and the joint manifestations of serum disease, have been recognized. It seems settled that acute rheumatic fever is always due to infection. This usually comes from lymphoid tissue and seldom from teeth. Other types of acute arthritis are usually also due to foci of infection, which are often around the teeth and gums.

Benjamin Rush, in 1801,[1] reported the cure of a patient with rheumatism of the hip by the extraction of a tooth. Many other clinicians have since emphasized the causal relation of dental infection to arthritis, especially of the chronic type, although opinions vary widely concerning the frequency with which dental infection occurs as the primary focus. Barker[2] thinks that the majority of cases of arthritis in middle life and in later life are due to dental infection. Wilcox[3] estimates that 90 per cent of all cases of chronic arthritis are due to dental foci. Cecil and Archer,[4] in studying 379 cases of chronic arthritis of probably infectious origin, decided that only 33 per cent were due to infection about the teeth.

As chronic diseases of the joints have

1. Rush, Benjamin: Medical Inquiries and Observations, Vol. 1, p. 199, Philadelphia; M. Carey and Son, 1818.

2. Barker, L. F.: Oral Sepsis and Internal Medicine, J. Dent. Res., 2:43 (March) 1920.

3. Wilcox, W. H.: Infective Arthritis and Allied Conditions; with Special Reference to Etiology and Treatment, Brit. M. J., 1:805 (June 4) 1921.

4. Cecil, R. L., and Archer, B. H.: Classification and Treatment of Chronic Arthritis, J.A.M.A., 87:741 (Sept. 4) 1926.

been more carefully studied and classified, it has become more and more apparent that many cases of arthritis formerly thought due to dental or other infection are in reality due to a disturbance of metabolism, errors in diet, abnormal absorption from the intestinal tract or to other toxemias not of bacterial origin. We are now much less prone to decide at once that every case of arthritis is due to infection. We still think that most cases of acute arthritis are the direct result of the metastasis of bacteria from a focus. Some cases of chronic arthritis are due to teeth. Probably no cases of true hypertrophic arthritis are due to focal infection. Chronic arthritis of the atrophic type may be due to focal infection, although it constantly assumes less importance in the treatment of the disease.

Diseases of the muscles, periarticular tissues, tendons and bursae are often associated with joint disease. Disorders of these tissues are often the result of dental or other focal infection, although it is possible that any factor in joint disease may also involve these tissues.

Most cases of arthritis due to infection result from the direct localization of bacteria in the joints. In a certain group of cases, the reaction in the joints is due to the absorption of toxins from the focus. I recall one case of acute arthritis in which removal of a badly infected tooth gave immediate relief from the joint symptoms. The next day after extraction, he could walk without trouble although previously he had been on crutches. Here, the joint symptoms must have been due to the presence of toxins or to allergy. The latter probably plays a more important part in infectious arthritis than we usually believe. The classic example of this type of allergic arthritis is that occurring in serum disease.

### DISEASES OF THE KIDNEY

One type of kidney disease, glomerulonephritis, is probably always of bacterial origin, the organisms entering from a focus of infection. The classic example is the glomerulonephritis occurring in scarlet fever in which the tonsils are the focus. Occasionally, dental infection may be the focus for such a lesion, for a nephrosis or for a chronic diffuse nephritis.

Disease of the kidney medulla and pelvis in the absence of obstruction to urinary flow is probably always due to hematogenous infection. There has been much discussion concerning so-called "pyelitis." The symptom complex which this term designates is very commonly met with in medical practice. The clinical picture of chills, fever, pain in the kidney region and pyuria is usually a pyelonephritis, with the principal lesion in the medulla of the kidney. In the past, these conditions have been ascribed to *B. coli* infection, although there is not much doubt concerning the etiologic relation of this organism to the disease. There is much to suggest that the initial kidney damage is due to the streptococcus or staphylococcus. Bumpus and Meisser[5] have shown experimentally that such kidney infections are frequently produced by bacteria brought to the kidney from areas of chronic dental infection. I have reported similar cases.[6]

Hunner emphasizes the relation of chronic infection to ureteral stricture and certain types of ulcer of the bladder.[7]

5. Bumpus, H. C., and Meisser, J. G.: Focal Infection and Selective Localization of Streptococci in Pyelonephritis, Arch. Int. Med., 27:326 (March) 1921.

6. Haden, R. L.: Relation of Chronic Foci of Infection to Kidney Infection, Am. J. M Sc., 169:407 (March) 1925.

7. Hunner, G. L.: End Results in Ureteral Stricture, J. Urol., 12:295 (Oct.) 1924.

Rosenow and Meisser have shown experimentally the possible relation of infection to renal calculi.[8] Rarely, other diseases of the genito-urinary tract, such as prostatitis, epididymitis, ovaritis and salpingitis, may result from dental or other focal infection.

The fact that focal infection may cause lithiasis and infection of the urinary tract, and possibly other diseases of the kidney such as nephrosis and chronic diffuse nephritis, seems definitely settled. Certainly, the elimination of infection in such conditions is always indicated.

### HEART AND VASCULAR DISEASE

Acute endocarditis is always due to some focus of infection. In young individuals, the focus is usually in the tonsils and adenoid tissue. In older individuals, endocarditis lenta, the type commonly encountered, is often due to dental infection. This is a very serious condition, usually progressive, and practically always ultimately fatal.

Myocarditis occurs very commonly in association with acute rheumatic fever. Chronic myocarditis is a frequent cause of death. Here, the heart muscle is being slowly injured over a long period of time, the muscle fibers being gradually replaced with fibrous tissue. Most observers believe that the initial damage is usually due to infection. This may occur during an acute infection such as typhoid fever or pneumonia. In many instances, the damage results from the continuous absorption of toxins over a long period of time from some chronic focus, which is frequently in the teeth. Some of the arhythmias which do not depend on permanent structural damage to the heart may be due to absorption from foci of infection. This is particularly true of transient acute auricular fibrillation and paroxysmal tachycardia.

There is much difference of opinion concerning the relation of chronic infection to arteriosclerosis and arterial hypertension of the essential type. It probably has little to do with either. Acute phlebitis and arteritis are often due to focal infection. I have reported several cases of arteritis in which I believe dental infection was probably the focus. The almost constant association of focal infection with thrombo-angiitis obliterans seems most significant.

### DISEASES OF THE ALIMENTARY TRACT

Digestive disturbances such as gaseous eructations and lack of appetite may result from the swallowing of products of infection about the teeth. These occur principally with pyorrhea. Most diseases of the alimentary tract due to dental infection are hematogenous and not due to swallowing infectious material.

There has been much discussion concerning the relation of focal infection to gastric and duodenal ulcer. Bevan[9] thinks that hematogenous septic infarcts of the mucosa may have a rôle in these ulcers but are not the common cause. Eusterman[10] states that the theory that infection is the cause of ulcer is admittedly the only tenable one at this stage of medical progress. Between those two diverse views is the classification of

8. Rosenow, E. C., and Meisser, J. G.: Production of Urinary Calculi by Devitalization and Infection of Teeth of Dogs with Streptococci from Cases of Nephrolithiasis, Arch. Int. Med., 31:807 (June) 1923.

9. Bevan, A. D.: Relative Value of Surgical and Medical Treatment of Gastric and Duodenal Ulcer, J.A.M.A., 79:22 (July 1) 1922.

10. Eusterman, G. B.: Recurrent Ulcer of Stomach and Duodenum, Minnesota Med., 6:698 (Dec.) 1923.

Smithies,[11] who divides ulcer from the standpoint of etiology into ten groups, and classifies one-third of the total number as bacterial.

Rosenow[12] has shown that the intravenous injection of streptococci may be followed by ulcer of the stomach and duodenum; that streptococci are commonly found in the ulcer of man, and that the injection of streptococci isolated from the ulcer wall as well as from foci of infection in patients suffering from peptic ulcer produces, when injected into animals, ulcers of the stomach and duodenum resembling those of man. Nakamura,[13] working in Rosenow's laboratory, injected sixty-six animals with cultures from the tonsils in cases of gastric ulcer and found gastroduodenal lesions in 70 per cent. Similar lesions were found in 25 per cent of control animals injected with tonsil cultures from other patients.

The results of such experiments afford evidence that focal infection may cause peptic ulcer. The frequent improvement resulting after removal of the focus is additional evidence. Another point is the anatomic location of the gastric and duodenal lesions in experimental animals. It is well recognized that gastric ulcer may occur in any part of the stomach but is much more frequent in the pyloric region. The experimental gastric lesions have a similar distribution. Clinically, duodenal ulcer is always limited to the first third of the duodenum. The usual explanation given for this localization is that only in this portion is free hydrochloric acid present, and that here the gastric contents are retained longer than in any other portion of the duodenum. In animals, the duodenal lesions are limited to the duodenal bulb.

Chronic ulcerative colitis has recently been added to the gastro-intestinal diseases probably due to focal infection. Bargen,[14] in an excellent series of experiments, has shown that this disease is due to a specific nonhemolytic streptococcus, which he has frequently recovered from chronic foci of infection, especially about the teeth. Marked clinical improvement has resulted from the elimination of the foci of infection and treatment with vaccines made from the causative agent.

Appendicitis or cholecystitis may result from dental infection. Acute dental infection may occasionally so metastasize. I am inclined to think that chronic cholecystitis is very infrequently due to focal infection. Barker[15] reports a case of hepatitis due to pyorrhea alveolaris.

Certainly the statement is justified that the importance of dental and other foci of infection in gastric and duodenal ulcer and in chronic ulcerative colitis is undoubted. The removal of foci in the treatment and prevention of such conditions is always indicated.

### DISEASES OF THE EYE

A close clinical relationship between oral sepsis and infections of the uveal

11. Smithies, Frank: Significance of Etiologic Factors in the Treatment of Peptic Ulcer, J.A.M.A., 74:1555 (June 5) 1920.

12. Rosenow, E. C.: Causation of Gastric and Duodenal Ulcer by Streptococci, J. Infec. Dis., 19: 333, 1916; Rosenow, E. C., and Sanford, A. H.: Bacteriology of Ulcer of the Stomach and Duodenum in Man, ibid., 17:219, 1915; Rosenow, E. C.: Specifity of Streptococcus of Gastric-Duodenal Ulcer and Certain Factors Determining Its Localization, ibid., 33:248 (Sept.) 1923.

13. Nakamura, T.: Focal Infection and Elective Localization in Ulcer of Stomach and in Arthritis, Ann. Surg., 76:29 (Jan.) 1924.

14. Bargen, J. A.: Etiology of Chronic Ulcerative Colitis, J.A.M.A., 83:332 (Aug. 2) 1924.

15. Barker, L. F.: Oral Sepsis and the Digestive Apparatus, Southern M. J., 11:481, 1918.

tract has long been recognized. Black,[16] in summarizing the literature concerning ocular disease resulting from dental lesions, cites clinical reports bearing on this relation which were published as early as 1842. Butler,[17] in a study of 100 cases of infection of the uveal tract, decided that 12 per cent were definitely due to oral sepsis. He considered that the etiology of 41 per cent was doubtful, but concluded that probably a large part of these originated in oral sepsis. B. T. Lang[18] classified seventy-one of 176 cases of eye infection as due to septic foci about the teeth. William Lang[19] thought that pyorrhea was responsible for 139 cases of 215 cases reported by him. De Schweinitz[20] aptly sums up the question in the statement that there is "no better established etiological relationship than that between septic foci in dental areas and certain diseases of the eye, notably those of the uveal tract." Irons, Brown, and Nadler[21] studied a streptococcus isolated from the infected tissues of a patient suffering from dacrocystitis and iritis. They found that a large percentage of rabbits developed eye lesions when injected intravenously with the freshly isolated culture. The ability to produce specific lesions was lost on subculture and on passage. The loss of a specific tendency to localize in the eye was not accompanied by any demonstrable decrease in virulence for rabbits. Rosenow[22] has reported cases of eye infection from dental pulpitis with the reproduction of the lesion in animals. I have reported a group of ocular disorders due to dental infection.[23]

16. Black, A. D.: Ocular Disorders Resulting from Dental Lesions, Ophth. Rec., 24, 1915.

17. Butler, T. H.: Etiology of Iritis, Brit. M. J., 1:804, 1911.

18. Lang, B. T.: Influence of Septic Infection in the Causation of Eye Disease, Brit. M. J., 1:381 (Feb. 22) 1913.

19. Lang, William: Influence of Chronic Sepsis on Eye Disease, Lancet, 1:1368 (May 17) 1913.

20. De Schweinitz, E.: Dental Infection in Its Relation to Ocular Disorders, Dent. Cosmos, 62:565 (May) 1920.

21. Irons, E. E.; Brown, E. V. L., and Nadler, W. H.: Localization of Streptococci in the Eye, J. Infec. Dis., 18:315, 1916.

### DISEASES OF THE BLOOD-FORMING ORGANS

Anemia is well organized as one of the cardinal symptoms of oral sepsis. The anemia is usually of the secondary type. Some clinicians believe that pernicious anemia also may result from focal infection. It is true that oral sepsis is exceedingly common in pernicious anemia, and the anemia is often improved temporarily by the removal of focal infection. The improvement is probably due to the removal of an added load rather than removal of the cause. I consider it exceedingly doubtful that pernicious anemia is ever due primarily to focal infection. At times, other blood diseases, such as hemophilia or chronic thrombopenia, may be due to dental or other focal infection.

### NERVOUS AND MENTAL DISEASES

Oral sepsis may be a factor in certain diseases of the nervous system. Trigeminal neuralgia, sciatica and other forms of neuritis are not infrequently due to dental infection. I have seen one case of encephalitis manifestly of dental origin. It is especially important to search carefully for infection of the dental pulp in cases of neuritis.

22. Rosenow, E. C.: Focal Infection and Elective Localization, M. Clin. N. America, 5:573 (Sept.) 1921.

23. Haden, R. L.: Elective Localization in the Eye of Bacteria from Infected Teeth, Arch. Int. Med., 32:828 (Dec.) 1923.

The symptom complex to which we give the name neurasthenia may be due to the slow absorption of poisons from a chronic focus of infection. Cotton[24] has studied especially the relation of dental infection to mental diseases and reported marked improvement after the removal of foci. Graves[25] has reported similar results. It is the opinion of many critical observers that the importance of focal infection is much overemphasized in such case reports.

## SUMMARY

There are few tissues of the human body in which bacteria from a dental focus may not localize. Clinically, only a small number are commonly found affected. The incidence of involvement as observed in clinical work checks closely with the incidence of lesions as found in the experimental animal after intravenous inoculation with organisms recovered from dental foci. The lesions most frequently seen in patients due to focal infection are those of the locomotor system. The kidney, heart, stomach and duodenum and eye are often affected. Less commonly, other organs, such as the nervous system and blood building tissues, may be involved. Rarely, very unusual lesions, such as onychia[26] or thyroid disease,[27] may result from dental infection.

Every clinician is disappointed in the results obtained by the removal of foci of infection in the treatment of disease even of proven metastatic origin. To me, the lesson to be learned from the demonstration of a definite relation of foci of infection to various systemic diseases is the need for the removal of such foci before metastasis to other organs has taken place. Disease, once initiated, is often not affected by removal of the cause. Why remove a cancer of the breast after metastatic nodules in the liver can be demonstrated? Why not remove foci of infection before metastatic disease has resulted? This is the lesson to be learned from all clinical and experimental demonstration of the causal rôle of chronic foci of infection in systemic disease.

## DISCUSSION

*Leon Bloch, Chicago, Ill.:* I remember as a young physician hearing the laity speak of convulsions from teething and of headaches during the course of eruption of third molars in young adults; and while we place little credence in the convulsion theory, we still see an occasional incident of headache attributed to impacted molars. This is of quite minor significance in comparison with the host of pathologic processes that ride in the wake of focal infection involving the teeth. The roentgen ray taught us a long time ago that a beautiful set of teeth or a fine piece of dental artistry is no guarantee against infection at the roots, unless such an examination has been made when there is reason to suspect the teeth and especially if other sources of infection have been ruled out. The severity of some of the results of focal infection, a fatal outcome in some instances or a lingering disability in others, must certainly arouse in us a fervent desire to continue the search to the very bottom. Nonspecific ulcerative colitis, as has already been pointed out, is one of the most baffling diseases that the physician has to contend with. There are few diseases in which the discouraging character of the course, the prolonged invalidism, the profound infection, in some cases, and the possibility of metastatic lesions and death are so pronounced as in ulcerative colitis. If it can really be shown that root abscesses are the determining etiologic factors in some

24. Cotton, H. A.: Treatment and Prevention of Certain Mental Disorders, New York State J. Med., 23:18 (Jan.) 1923.

25. Graves, T. C.: Focal Infection as a Factor in the Production of Mental Disorder, Proc. Roy. Soc. Med., 19:16, 1926.

26. Haden, R. L., and Jordan, W. H.: Multiple Onychia as a Manifestation of Focal Infection, Arch. Dermat. & Syph., 8:31 (July) 1923.

27. Bohan, P. T.: Ligneous Thyroiditis, Associated with High Grade Dental Infection, M. Clin. N. America, 7:1069 (Jan.) 1924.

cases, as Bargen has suggested, we have in the eradication of this focus a form of prevention and treatment of incalculable value. Acute endocarditis is another bugbear to the doctor, because, once started, we do not know where it will end. I can cite from my own experience instances of such infections improving after the removal of abscessed teeth. If not fraught with such disastrous consequences, the disabling character of chronic arthritis in a way presents almost as gloomy a picture as do the instances of infection with fatal consequences. Fortunately, the monarticular type of arthritis is the commonest form of arthritis resulting from dental infection and more than any other form is favorably influenced by the removal of the focus of infection. Dr. Haden has told us that chronic glomerulonephritis is occasionally the result of dental infection. Several years ago, Dr. Hektoen found minute infarcts in the kidneys of rabbits which he had injected with various bacterial vaccines. It is well known that perinephritis occurs as a complication of tonsillitis. I cannot recount any instance of this condition resulting from dental infection. It is necessary to rule out the teeth as a source of trouble if no other focus of infection is found. I agree with Dr. Haden, as a result of my own experience, that dental infection can rarely if ever be laid at the door of cholecystitis or appendicitis. Much more frequently, the appendix assumes the rôle of focus of infection. Dr. Haden has called attention to a form of neurasthenia in which a most complete and searching examination may reveal nothing but a secondary anemia. Still, the patient has well-defined complaints, early fatigability, even after very slight exertion, inability to concentrate and a condition bordering on invalidism. A roentgen-ray examination of the teeth may reveal numerous root abscesses. Extirpation of the teeth involved and thorough treatment of the abscess cavities lead to a complete recovery of the patient. On several occasions, I have had patients with choroiditis and retinitis referred to me by oculists, with instructions to search for the cause. The commoner causes, such as diabetes and nephritis, were well known to me. I have learned from my contact with the oculists not to fail to have a thorough dental examination made because, in not a few instances, root abscesses were disclosed as the cause. Attention to this source of trouble frequently resulted in an almost complete recovery of the eye condition. This aspect of the rôle of focal infection is very well known to the oculist and a search in the teeth for the noxae should be the rule in every case. As regards diabetes, a carefully followed dietary régime, with or without the aid of insulin, will meet with happy results in almost every case. Occasionally, we meet with cases of diabetes that seem to be refractory to insulin unless it is given in exceedingly large doses. It is well known that, in the presence of an acute infection, the loose rule of administering one unit of insulin for every 2 gm. of carbohydrate over and above the patient's tolerance does not hold. I wish to cite an instance of a child of 13 with diabetes who had been receiving fairly large doses of insulin before every meal. Some time after I saw her, she developed a toothache. A roentgen-ray examination of the teeth revealed an infection not only at the root of the tooth that was aching but at the roots of several others which were not suspected of being involved. A removal of all the offending teeth not only led to an improvement in her general condition but also the use of insulin became unnecessary in the subsequent course of her treatment. I call particular attention to this as an example of dental infection where least expected. Unfortunately, eradication of dental infection is not always blessed with brilliant therapeutic results. I am sorry to say that the unsuccessful result of the removal of foci of infection has led some competent internists to be skeptical regarding the importance of the rôle of foci of infection in the causation of some diseases. A secondary focus of infection may be established in a metastatic lesion. Hence, the apparent futility of the removal of the original focus. It is on this account that one must agree with Dr. Hayden that preventive treatment should be instituted much earlier than it is. I am not in favor of general extraction of all teeth that are devitalized nor of the indiscriminate removal of all the teeth for pyorrhea as is sometimes done, but I do advocate a search for infection and discriminating removal of teeth which harbor infection at their roots. Especially should such an examination be made in every instance of vague and indefinite complaints where no apparent cause for the disability has been found. By such a procedure only can we hope to prevent or defer troublesome consequences.

*C. F. B. Stowell, Chicago, Ill.:* The focal infection theory is passing through a period

of transition and will soon become an admitted scientific fact or law along with gravitation and evolution. Like Darwin and Newton and Copernicus in their work of advancing human knowledge, the men who have done the research in, and the announcement of the findings in connection with, foci of infections have been anathematized; but such has always been the reward of those who have been instrumental in unseating harmful traditions. As grave as I consider the potentialities of foci of infections, I have never excluded from my mind the possibilities of their presence in other tissues of the body as well as those of the teeth. At the same time, I have always had a lively realization that the tissues of the teeth are within my province as a dentist and that I therefore had accepted a great responsibility. If a person becomes sick and the condition does not respond readily and favorably to the usual empiric treatment, the physician of today begins to think of foci. He orders roentgen-ray examinations of the teeth, and, if thorough, he tries to find other possible foci. There is no argument in connection with the advisability of removing operable foci. The finding of a focus may be easy; but it is quite difficult at times to find the focus of distribution causing a particular lesion. Any focus is a menace and an overload. The removal of dental foci, when they are not deemed the direct cause, may raise the patient to a plane of health that will enable him to stand better the shock of more serious advisable operations. The dentist is, generally speaking, in a more advantageous position as regards prophylaxis than is the physician. The latter in comparison has but little chance to prevent disease, in the single case. The dentist, on the other hand, by his intimate knowledge of his patient's teeth and his knowledge that devitalized teeth constitute one of the great centers of infection, can prevent much systemic disease, if he practices in accord with this concept. We can prevent just about 100 per cent of all acute and chronic apical abscesses and their sequelae — cysts, other local resultants, and the various metastatic diseases attributed to dental infectious foci by Dr. Haden and other research men. Regardful as I am of the technical skill that many have attained in what has been miscalled root-canal surgery and of that valuable auxiliary in diagnoses, the roentgen-ray, I am still very skeptical that anybody can produce convincing, absolute and scientific evidence concerning the presence or absence of infection in the apex and the periapical tissues of a devitalized tooth *in vivo*, and therefore we cannot assure the host of such a tooth that its retention is for his ultimate welfare. U. G. Rickert has described for this purpose a method of examination of the apical space. A surgeon, a bacteriologist and a technical laboratory equipment is required. Any usefulness it has is limited to the six upper anterior teeth, and even then the material obtained for examination would in all probability be useful but momentarily as evidence. We have been working on wrong hypotheses. We have assumed that there is a special dental pathology and therefore special dental therapeutics. We have assumed that there has been granted a special dispensation and that through some hocus pocus or legerdemain dental tissues act differently in cases of infection from other tissues of the body, and all because we were taught dogmatically. If dental students of fifteen or more years ago had been taught as I believe they are now that all scientific subjects are in a state of flux and that the opinions of today might be quite obsolete tomorrow, their minds would be more open to new concepts. If we have a devitalized tooth under consideration and there is an absence of subjective and objective symptoms, pain, swelling and redness, we are apt to pass the case as noninfected, especially if the roentgenogram discloses no structural changes in the investing tissues and tooth apex. If a series of roentgenograms have been made periodically, the first of the series showing considerable rarefaction, the second showing less and so on to the vanishing point, we are prone to pronounce the apical conditions as returned to normal and as being noninfected, though it is quite well known and accepted that bone repair can and often does take place in the presence of infection. Dr. Haden and others have had such teeth extracted, obtained from them positive cultures of streptococci and therewith produced in rabbits the patient's lesions. The retention of such teeth in the jaws is highly experimental. Too large a percentage of devitalized teeth appearing negative in the roentenogram have been proved infected. Our vaunted prevention program must have a greater margin of safety than that afforded by the devitalized tooth. Why wait until the patient has become

hospitalized before we grudgingly become willing to render our best oral health service? Let altruism take the place of a part of our egoism and egotism. It is a matter of great regret that the pulp became involved; but why gamble with the probability of greater calamities: the lesions of the hearts of young people and middle aged patients and the long list of degenerative systemic diseases demonstrably caused by dental foci? Let us get another view of human teeth and their economic importance. They are highly specialized organs. The environment for which they were specialized passed ages ago, long before political and social history commenced. To paraphrase Dr. Dorsey, our dentition is as well adapted for spinach as for beefsteak, specialized for neither, nor for cooked foods. At best, our incisors are only fair cutters, yet they were specialized for just that; our bicuspids are not strong enough to crunch bones, but they once were; our molars are not very good millstones, yet that is what the prehistoric man used them for. Modern human teeth are in a great many senses vestiges, and like other vestigil organs, such as the tonsils and the appendix, they are more liable to become the seat of infectious foci than other organs and tissues that at present enjoy the peak of usefulness. We have prepared ourselves as dentists to try to stay and to repair the ravages of an evolutionary movement that seemingly includes the teeth. It is a herculean task. Whether or not the race becomes edentulous eventually is unimportant. As a profession, we shall meet and adapt ourselves and our methods of practice to our problems as they are presented. In the meantime, let us protect our present-day public from the systemic diseases that have come, or, in all probability, may come, from pathologic tissue conditions within our province; remembering all the time that the replacement of teeth is a possibility, while this is not true of heart-valves, etc. Cures, as Dr. Haden has pointed out in the most significant paragraph in his essay, are rare, and let me add that they are rarer still in partial elimination of foci such as result from attempts to differentiate on a basis of magnitude of roentgenographic evidence which teeth to remove and which not to remove. Partial operations clearly defeat the objects of surgical interference and tend to discredit the focal infection theory. Furthermore, we cannot predetermine the time when a dental infection will affect a metastasis, nor the tissue or organ it will elect for its toxic products or colonization. In our dental examinations of roentgen-ray films, we too often hear it said, "This tooth has been treated (mental reservation, meaning devitalized), but it doesn't show much that is wrong." We hear the dentist say to the patient, "You are in perfect health, why worry? Let us have this tooth x-rayed from time to time to keep track of it." This attitude is worse than unscientific. In this connection, I have always taken pleasure in quoting from the writings of one of the most genuine research men of the dental profession, the late Clarence J. Grieves. Dr. Grieves states the matter thus: "The dentist who without medical assistance of the highest order attempts to distinguish between the sick and the well patient, and who presumes by a glance and a few routine questions to determine instantly matters which might keep hospital laboratories busy for days, dabbles superficially in internal medicine, when he does not know the actual etiology of, nor the sure cure for, a single dental disease." Yet we presume to say when these local pathologic conditions are causing systemic disease and when not. Would it not be better to be a bit more modest and give heed to the leaders of the medical profession, especially when local pathologic conditions affect tissues and organs out of dental bounds?

# MAINTENANCE OF SPACE AFTER EXTRACTION AND TREATMENT OF MALOCCLUSION OF THE DECIDUOUS TEETH AS A PREVENTIVE MEASURE*

By WILLIAM R. HUMPHREY, D.D.S., Denver, Colo.

THE desire to prevent deformity and to create conditions in the mouth favorable to normal growth should be the ambition of all dentists. Malocclusion, caries and loss of the permanent teeth all present problems which tax the efforts of the dental profession to such an extent that little effort has been expended to prevent these conditions. Notwithstanding all the progress made in dentistry during the past twenty-five years, including the marked improvement in dental education, caries of the teeth is still one of the most prevalent diseases of childhood.

The condition discovered in cities where regular dental inspection of public school children has been adopted is enough to challenge the dental profession to give less time to controversial papers on restoration and more thought to prevention. W. J. McMenamy, dental examiner in the Denver public schools, furnishes me with data covering the examination of 30,000 children, 96 per cent of whom have dental defects, and 60 per cent of whom have malocclusion of the teeth; that is, a total of 18,000 children out of 30,000 have malocclusion. A similar condition is found in all the cities of this country where like surveys have been made, with which all of you are familiar.

Dental clinics have been established in public schools to care for children whose parents cannot afford to pay for dental service, and those who can pay have been urged, and in some instances ordered, by the school authorities to have dental work done. Now let us assume that this system is carried out to complete satisfaction; all necessary restorations are made, and infected teeth are removed. The end results obtained under this plan, now being followed, present too high a percentage of malocclusion for us to maintain that decided improvement should not be made.

The matter of space retention as a preventive of malocclusion has received attention. Papers have been read before the American Dental Association, orthodontic societies and also the Society for the Promotion of Children's Dentistry, urging the retention of space after extraction of deciduous teeth. But judging from the large number of cases presented to orthodontists for treatment which are caused or complicated by this condition, it seems prudent for the dental profession and orthodontists in particular to advocate more attention to preschool dentistry. The lay public is ready for this idea of treatment, and all dentists should awaken to their responsibility in the treatment of these children.

Susan M. Coffin said before the Section on Children's Dentistry, Massa-

*Read before the Section on Orthodontia at the Midwinter Clinic of the Chicago Dental Society, Jan. 14, 1930.

chusetts State Dental Society, "We examined recently in our Demonstration Well Child Conference 851 'well' children who were 'all ready' to enter school according to their mothers or the neighbors of these children; 86 per cent had definite physical defects needing medical attention. Incidentally, 51 per cent had dental defects, and these were all children under 6 years of age."

In this day of slogans, it would be well not only to repeat in dental societies and quote in dental journals the importance of taking the child to the dentist for examination at least as early as at 3 years of age; but also to imbue the public mind

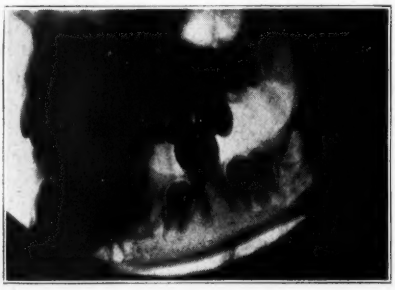

Fig. 1.—Premature loss of mandibular right second deciduous molar.

with this important fact by adopting a national slogan urging dental examination and instruction in oral hygiene as soon as the deciduous teeth erupt.

Most progressive dentists have adopted as routine practice roentgenographic examination of adult patients for initial caries, but few have thought to extend this service to children. I believe it is an irrefutable fact that the normal functions of the deciduous teeth are more important to the child than are those of their permanent successors to the adult, not only in mastication but as factors in normal development.

Until research has established a means for the prevention of caries, and since experience has taught us the difficulty of retaining large restorations in deciduous teeth without the loss of vitality of the pulp, and consequent loss of the tooth, we must, in the interest of a reduction in malocclusion, prevent this condition or its bad results. The first suggestion in this regard is to examine children of preschool age in order that incipient caries may be discovered and stopped. The second suggestion is to retain space where deciduous molars must be extracted prematurely. The third suggestion is to correct the cases caused by premature loss of teeth as soon as they are discovered by placing the teeth in positions of mechanical advantage and retaining the space so created. The fourth suggestion is to treat mesial and distal occlusion in the deciduous dentition before the eruption of the first permanent molar. The fifth suggestion is for all dentists to assist in dental health education. This last, of course, covers many phases of the situation, beside the point in this discussion.

Now, the question arises: In what percentage of cases of malocclusion is it possible to intervene and create conditions favorable to normal growth? Undoubtedly, the percentage is high. The comparatively few cases of mesial and distal occlusion in the deciduous dentition and the high percentage of cases which show the effects of premature loss of space in the deciduous dentition lead me to believe that many of the difficult cases which reach us after all the permanent teeth have erupted, in which the etiology has not been clearly understood, can be traced, in part, to the premature loss of deciduous teeth. At least half of the cases of malocclusion now seen could be checked and growth diverted toward

the normal, if the procedures just described were observed.

The loss of space is so serious that even the mesiodistal width of deciduous teeth plays an important part in development. Noyes in his "Dental Histology," chapter 27, states: "While the growing tooth germs are producing force which causes conditions of stress of the cortical plates, the growth of the tissues within the mouth, the tongue and the associated organs, is exerting pressure on the lingual surface of the bone. The muscles attached to their surfaces transmit force to the bone through the periosteum, and the functions of mastication, deglutition and respiration are acting upon them. All of these are mechanical stimuli, to which the connective tissue cells respond. . . . Any malocclusion disturbs the balance in distribution of forces, and results in a disturbance of the development of bones."

While I believe that clinical data will substantiate this statement, it is well to consider some other factors which might tend to contradict this theory of development. Northcroft reports two congenitally edentulous cases and states that the jaw growth was practically normal. Many others have reported cases of teeth congenitally absent in which growth has been apparently normal. But clinical experience in our practice, where we have not only the casts to examine but the face and skull as well, shows us that a lack of normal development can invariably be detected in cases not in normal function.

We have all found impacted bicuspids in cases in which the deciduous teeth have been in normal function and which may even have been retained so long as to require forcible removal. We also find maxillary permanent cuspids outside the dental arch where the deciduous teeth have been retained in function. These cases, of course, are rare as compared to the many cases of malocclusion apparently caused by the premature loss of deciduous teeth. The cause of this lack of local development has not been explained satisfactorily, but it may have etiologic factors which are of dietary

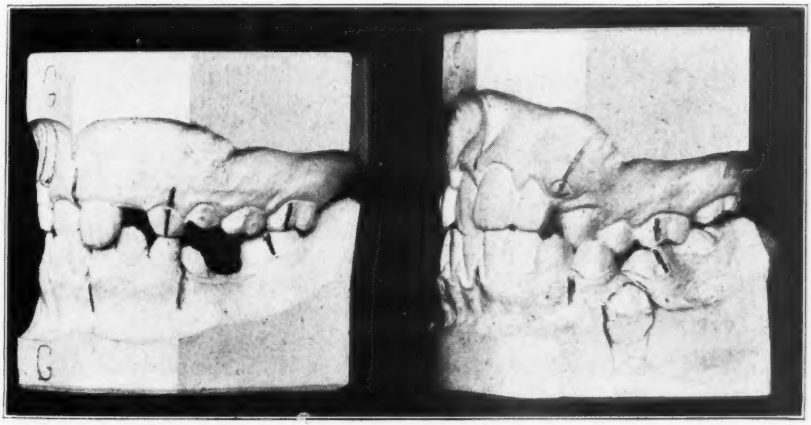

Fig. 2.—Two cases showing result of premature loss of mandibular left second deciduous molar. Left: Cast from a patient, aged 9 years; right, from a patient, aged 12 years.

origin, or they may be symptoms of disease of the endocrine system.

The field of endocrinology beckons just now, and it is becoming increasingly common to hear dentists refer to endocrine disturbance. No doubt, many cases of malocclusion are caused by disturbed endocrine function. However, these facts are only added reason for the maintenance of space in the deciduous dentition, because, if malocclusion can and does develop from premature loss of deciduous teeth in children when the growth rate is average, it most certainly will and does develop to a more serious degree when the osseous development is not average.

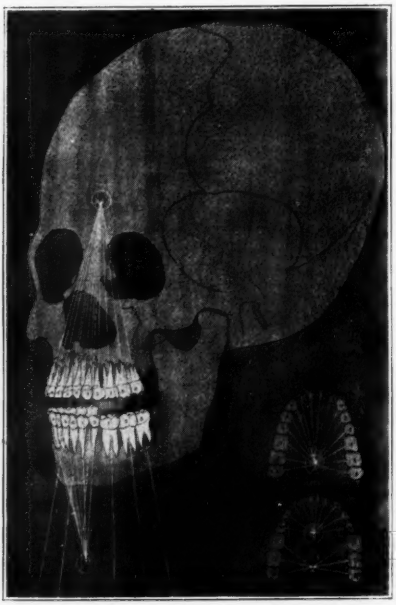

Fig. 3.—Isometric drawing showing the teeth together with lines of their long axes inserted in the skull.

The first thing to consider in the maintenance of space is maintenance of the mesiodistal diameter of the deciduous teeth. The loss of the mesiodistal diameter of the deciduous teeth often produces malocclusion, and a subsequent lack of development, even though there is no loss of teeth.

Many of us have observed clinically that ankylosed jaws do not grow normally, and it is quite generally accepted that function of the tongue and associated organs, and the muscles attached to their surfaces, is the most important factor in the development of the jaws. Through the loss of the mesiodistal diameter from interproximal decay, the force of the normal approximal contact is lost, which causes a mechanical impediment to normal function and a disturbance of the development of bone.

The premature loss of either the first or second deciduous molars, now so common, presents problems in space maintenance of such importance as to merit a careful analysis. We will first consider the clinical picture presented by the premature loss of a mandibular second deciduous molar. All our practices are filled with cases caused or complicated by this condition. (Fig. 1.)

Todd states, "Growth consists of two processes, addition of substance and abstraction of substance. Bone is constantly changing in its modeling. We must not be misled by the hardness of bone into thinking of it as something permanent like marble or cement. It is a living tissue subject to constant change. It undergoes processes of metabolism like other tissues of the body. So whenever we consider growth we must not regard it as purely increase in dimension, we must think of it also as a process of remodeling." This remodeling process takes place after the premature loss of deciduous teeth, and the earlier the tooth is lost, the greater and the more unfavorable the change.

Figure 2 shows the models of two cases; left, in a patient aged 9 years, following loss of a mandibular second deciduous molar; right, in a patient aged 12 years, showing increasing deformity. The permanent molar usually grows forward and toward the lingual aspect. Occasionally, it is deflected buccally. In either event, it frequently produces an impaction of the second bicuspid. There is not only the cusp change in cases of this nature to consider, but also the rotation and change of the long axes of the permanent molars. This change of axes, a change in remodeling of bone, is of much more importance than the cusp interference presented in such cases, not only because of the difficulty encountered in treatment but also because of the influence of the change that this first

Fig. 4.—Two cases showing result of premature loss of right maxillary second deciduous molars and forward growth of maxillary first permanent molars.

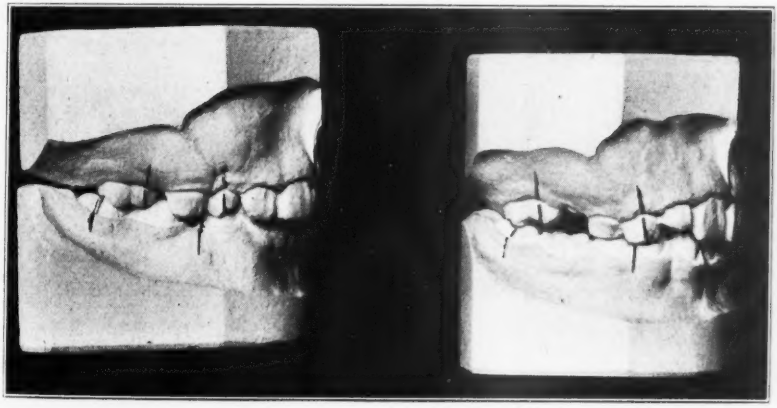

Fig. 5.—Two cases showing result of premature loss of right maxillary second deciduous molars and forward growth of maxillary first permanent molars; buccal view of cases shown in Figure 4.

tooth undergoes on changing the long axis of each succeeding tooth as it erupts. In the normal occlusion, there exists not only the most advantageous relationship of cusp ridge and embrasure contact, but also a definite position of the long axis.

Figure 3 is an isometric drawing similar to the ones used by Monson. This has nothing to do with the spherical theory of occlusion, but is drawn to show the teeth together with lines of their long axes inserted in the skull. Great care was exercised to project the angles, and I am convinced that they are reasonably correct. I desire only to present the hypothesis that for each normal occlusion there exists a dimensional norm not often considered in discussions of occlusion. In cases in which there has been the premature loss of deciduous teeth and the subsequent change of the inclination of the long axes, this inclination requires correction before any hope of a satisfactory result can be realized.

Figures 4 and 5 show two cases of the premature loss of a maxillary deciduous molar with the subsequent forward growth and change of axes of the maxillary first permanent molars. It requires but the eruption of the remaining permanent teeth to bring on either a unilateral distal case (Fig. 6) or a case in which both maxillary second deciduous molars are prematurely lost, with the subsequent forward growth of the maxillary first permanent molars, often pro-

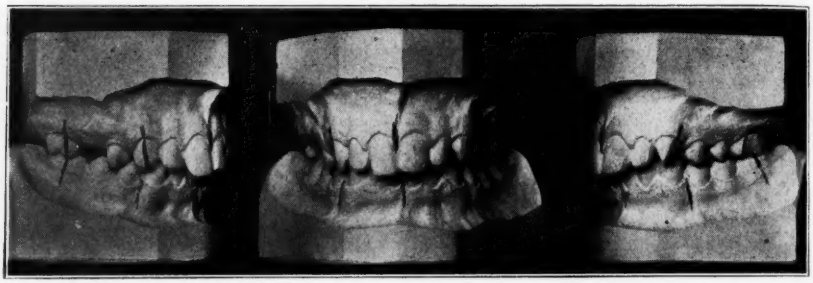

Fig. 6.—Case of type often diagnosed as unilateral distoclusion; caused by forward growth of maxillary posterior teeth on left side after premature loss of maxillary deciduous molar.

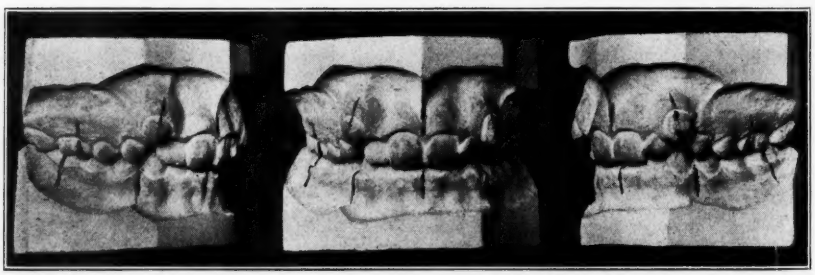

Fig. 7.—Malocclusion caused by forward growth of maxillary permanent teeth after premature loss of deciduous teeth.

ducing the classic example with which we are all familiar. (Fig. 7.)

The premature loss of the maxillary deciduous anterior teeth often causes a mesial occlusion, brought on by the mandibular incisors erupting first labially from the upper ridge, and the maxillary teeth erupting later to a position lingually from the mandibular incisors. (Fig. 8.) The figure on the left also shows to what extent a deformity can go from the premature loss of practically all the deciduous teeth, due both to dietary and dental neglect. Figure 9 shows the occlusal views of this case, which presents practically a complete impaction of the permanent teeth. The mucous membrane had become toughened and had to be removed surgically to assist the eruption of the teeth.

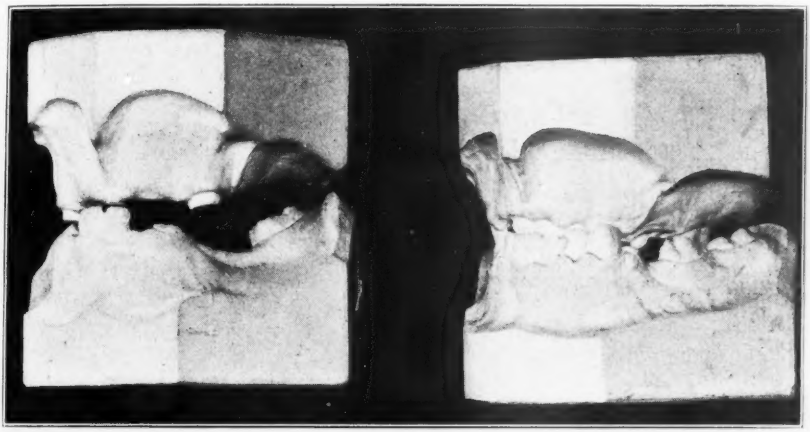

Fig. 8.—Mesial occlusion caused by mandibular incisors erupting first labially from upper ridge and maxillary teeth erupting later to a position lingually from mandibular incisors.

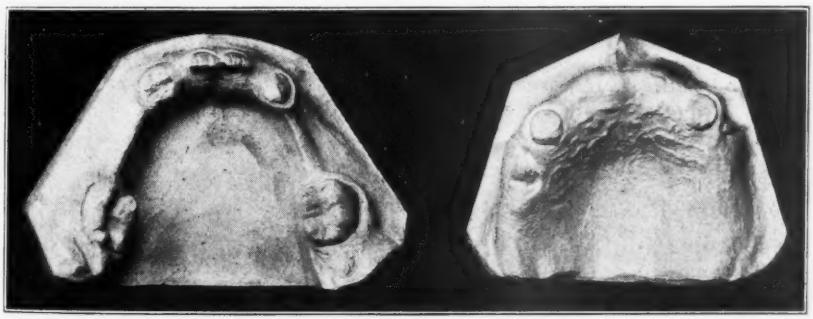

Fig. 9.—Occlusal view of case shown on left in Figure 8, which presents practically a complete impaction of permanent teeth.

Many methods of treatment have been suggested to overcome these conditions,

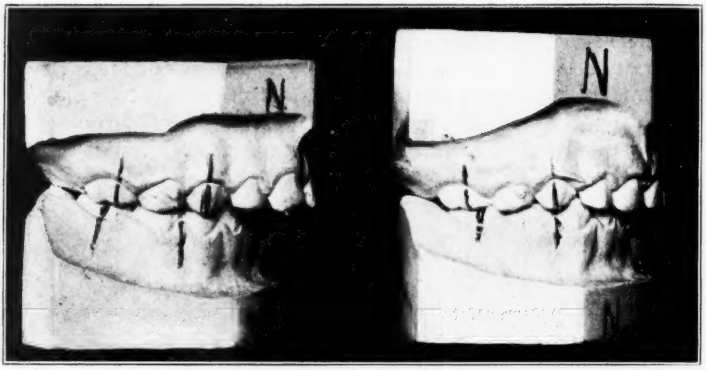


Fig. 10.—Models showing posterior teeth on right side of a distoclusion case, in a patient aged 3 years and 10 months. Right: Result of eleven months' treatment.

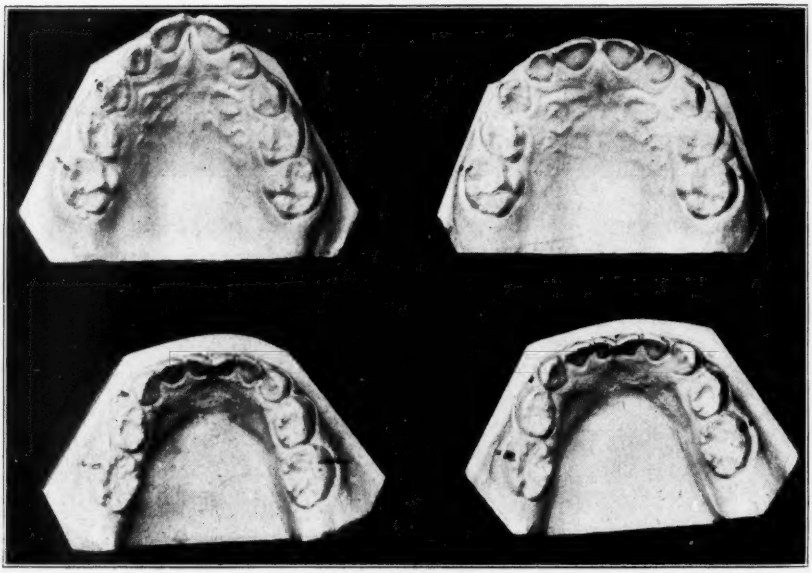

Fig. 11.—Case shown in Figure 10, occlusal view.

and to say that it could not be done would be to refute much of the evidence collected by orthodontists during the past thirty years, but prevention of many difficult cases is possible. Since it has been impracticable to establish orthodontic clinics to care for these advanced cases, few but those able to pay the fees of a private practitioner are receiving attention.

Albert H. Ketcham has, for many years, advocated the early treatment of mesial and distal occlusion of the deciduous teeth, and having had the privilege for the past ten years of observing his cases as long as from twenty to twenty-five years after treatment, I have come to the conclusion that such cases cannot be treated too early, and it is our practice to treat them before the eruption of the first permanent molars; in some instances, at between 3 and 4 years of age.

I do not believe a discourse on prevention is complete without the inclusion of treatment of these conditions. I therefore present first a distal occlusion case in a girl, aged 3 years and 10 months. The cause was thumb sucking. Figure 10 shows the occlusion before treatment, on the left, and after treatment, on the right. The treatment consisted of expanding the maxillary arch by means of a 0.03 round arch attached to the second deciduous molars by round buccal tubes,

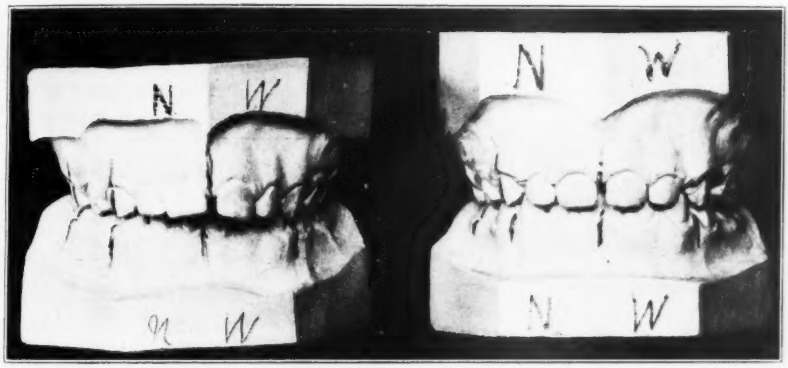

Fig. 12.—Case shown in Fig. 10, anterior view.

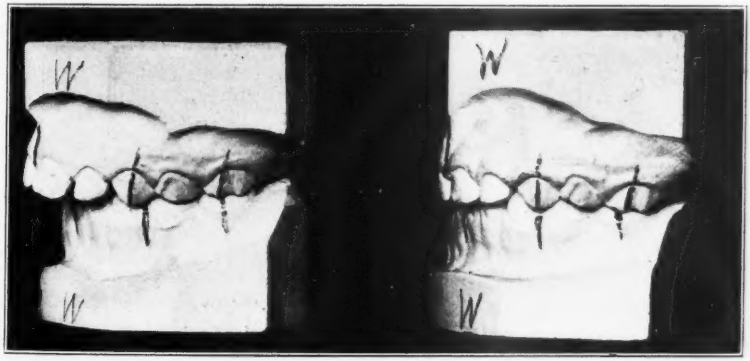

Fig. 13.—Case shown in Figure 10, left side.

and to the deciduous cuspids by the Ketcham hook. The anterior portion of the arch was bent into a cupid's bow and gradually enlarged to the shape of Figure 11, right, the model on the left showing the condition before treatment. No appliance was used on the mandibular teeth, but the child was instructed to place her teeth in positions of mechanical advantage and to masticate in that position, the treatment suggested by A. P. Rogers, of Boston. Figures 12 and 13 show other views of the models. The models on the right of each illustration show the result of eleven months' treatment. Figures 14 and 15 show the face; left, before treatment, and right, two and one-half years after treatment. The patient has had no subsequent treatment, but no doubt will require rotation of some of the teeth and should be under observation during the eruption of the permanent teeth.

The second case (Fig. 16), in a girl, aged 5 years, was a mesial occlusion, cause not known. The history revealed chronic tonsillitis during childhood. The mother first noticed that the mandibular incisors bit labially from the maxillary incisors at the age of 10 months. She consulted a dentist and was told to wait until the child lost the deciduous teeth and the permanent teeth erupted because any care given would have to be repeated if the case was treated early. The treatment consisted of the correction of the mesiodistal relation and some expansion of both mandibular and maxillary arches. (Fig. 17.) A lingual arch was used on the maxillary arch with hooks attached to the maxillary deciduous molars for the attachment of intermaxillary liga-

Fig. 14.—Left: Appearance of patient before treatment. Right: Appearance two and one-half years after treatment (case shown in Figures 10-13).

tures, and a 0.030 labial arch was used on the mandibular teeth and attached to the mandibular second deciduous molars by means of round buccal tubes, and to the deciduous cuspids by means of Ketcham hooks, the anterior section bent in the form of a cupid's bow. Hooks for intermaxillary ligatures were attached to a point just forward from the mandibular cuspids. Figure 18, left, shows the case before treatment; right, after thirteen months' treatment. Figure 19 shows the buccal views of the right side of the jaws. Figures 20 and 21, left, show the face before treatment; right, after treatment.

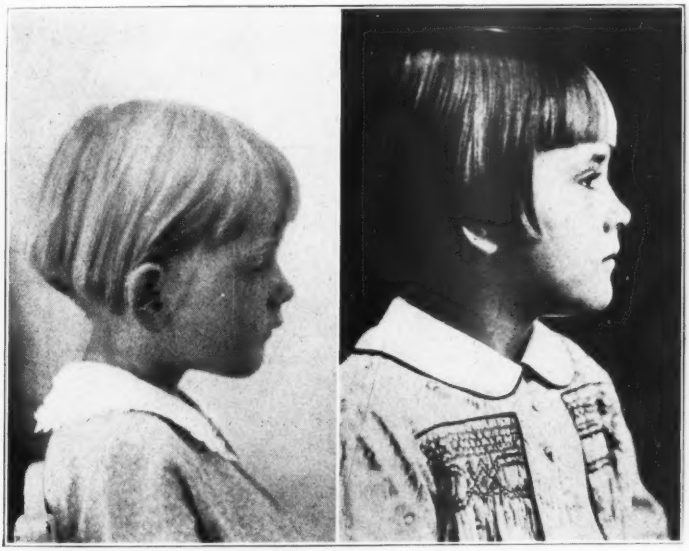

Fig. 15.—Profile of case shown in Figures 10-14.

## CONCLUSION AND SUMMARY

Children of preschool age are susceptible to tooth caries.

If caries runs rampant, loss of the mesiodistal diameters of the deciduous teeth or the loss of the teeth themselves results.

The loss of the mesiodistal diameters of the deciduous teeth, or perchance of the teeth themselves, results in disturbances of jaw development and of cuspal relationship of the permanent teeth. But, more than this, it changes the planes of the long axes of these permanent teeth, an injury that may never be rectified.

When necessity requires the removal of deciduous teeth, a simple space retainer should be made immediately and the case kept under observation.

Mesial and distal malocclusion of the deciduous dentition should be treated before the eruption of the first permanent molars and the deciduous teeth placed in positions of mechanical advantage.

These and other profound disturbances of childhood may be largely circumvented

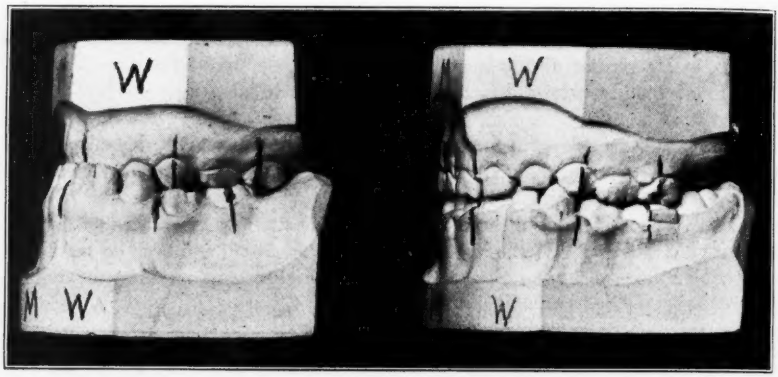


Fig. 16.—Models showing posterior teeth on left side of mesioclusion case, in a patient aged 5 years. Right: Result of thirteen months' treatment.

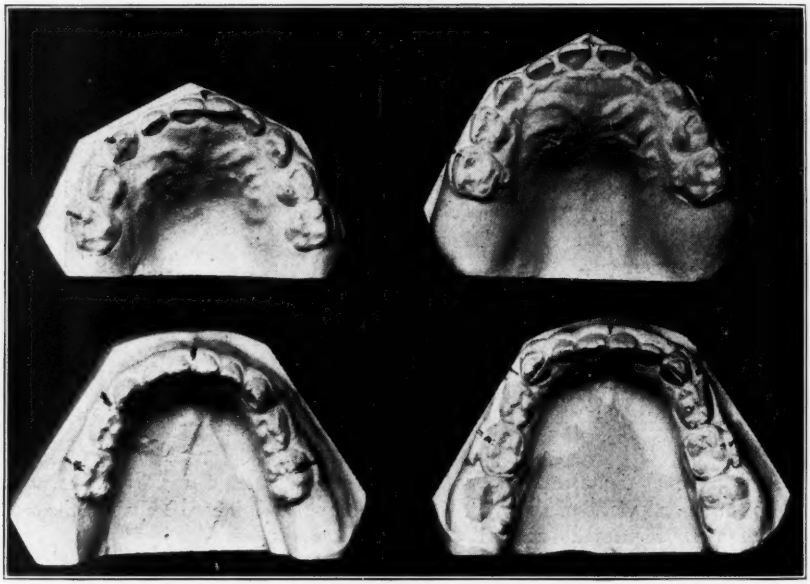

Fig. 17.—Case shown in Figure 16, occlusal view.

if we unite in a determined effort to have the mouths of preschool children carefully and systematically examined.

1232 Republic Building.

DISCUSSION

*F. E. Haberle, Chicago, Ill.:* In any discussion of the causes of malocclusion, the premature loss of deciduous teeth has always

played a most important part. That much time and effort have been given to a study of this problem and to that of the proper maintenance of space following such loss is evident on glancing through the recent orthodontic literature. Preventive dentistry is the keynote of the present day. By an adoption of the five suggestions presented by Dr. Humphrey for a reduction in the high per-

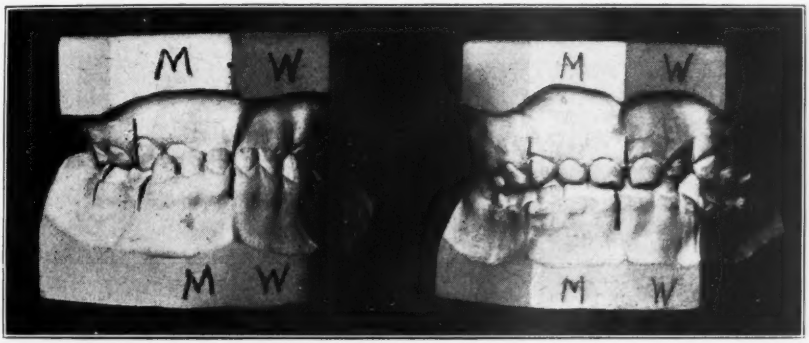


Fig. 18.—Case shown in Figure 16, anterior view.

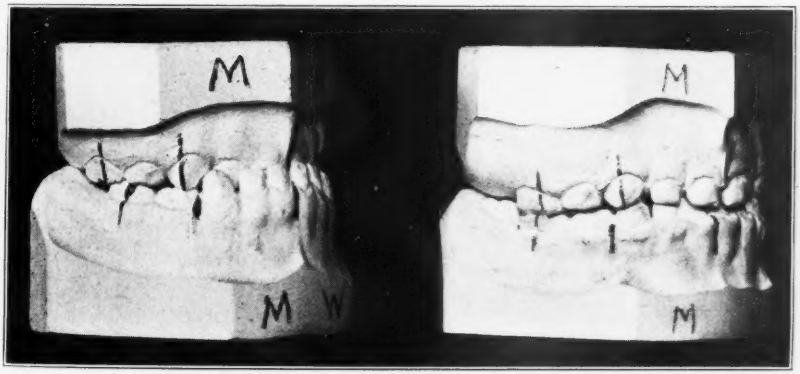


Fig. 19.—Case shown in Figure 16, right side.

centage of malocclusion now existing, a forward step in true prevention would be taken. A better knowledge of normal occlusion and the ability to recognize malocclusion by the profession as a whole might well be stressed as an aid in carrying out his suggestions. Through this better knowledge and ability, much good would come, and many incipient cases of malocclusion would be checked at an early stage in their development. Dr. Humphrey has laid down the principle that malocclusion should be corrected as soon as it is discovered. Earlier discovery and treatment would surely greatly reduce the large number of "horrible examples" of malocclusion, many of which are beyond hope of correction. The archaic and unfortunately poor advice, "Let things alone until all the permanent teeth are present," and "Wait and Nature will take care of it," is still being given. This makes for a most embarrassing situation after treatment is advised in its early stages. Early treatment has long been advised and prac-

Fig. 20.—Left: Appearance of patient before treatment. Right: Appearance thirteen months after (case shown in Figures 16-19).

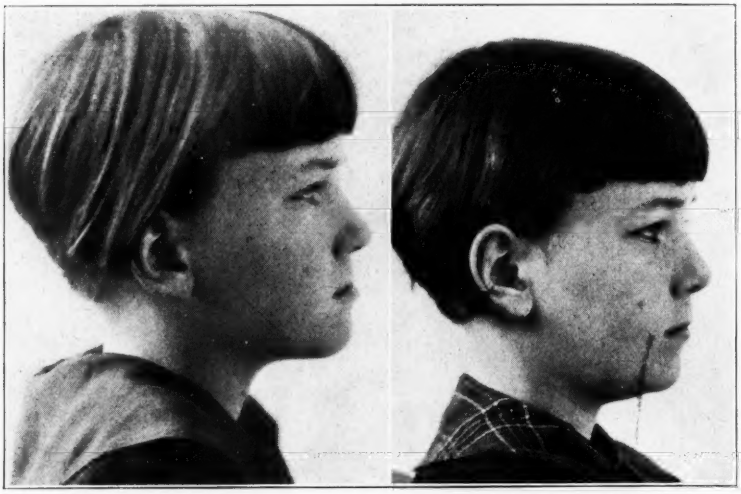

Fig. 21.—Profile of case shown in Figures 16-20.

ticed in the correction of distoclusion and mesioclusion cases in deciduous dentures. The necessity for proper maintenance of space after the extraction of a deciduous tooth is readily seen from the visual evidence presented by Dr. Humphrey. What the result of the loss of any given tooth may be is a matter of conjecture. In an investigation of the loss

of a single tooth, the first permanent molar, B. O. Sippy found any number of possible variations from the normal. Among the forces holding a tooth in position are the proximal contacts of the adjacent teeth in the same arch and occlusal contact of the tooth or teeth in the opposite arch. To maintain the proximal contact through the use of a mechanical device is not so difficult as is the maintenance of occlusal contact. Where there is multiple loss of teeth, considerable difficulty may be encountered in preventing elongation of the opposing teeth. The space is so long that a fixed space maintainer is liable to be bitten and distorted out of position. The advisability of covering the space with a removable vulcanite device might be questioned. A space maintainer to best perform its function must (1) maintain the mesiodistal diameter of the deciduous tooth lost, not that of the permanent successor; (2) be so constructed as to allow for future normal growth and development; (3) allow for proper cleaning, and (4) make some provision for the normal movement of the abutment teeth, i.e., must furnish some form of stressbreaker.

# ETIOLOGIC FACTORS IN VINCENT'S INFECTION*

By FRANCIS H. DALEY, D.M.D., Boston, Mass.

THE gingival tissues, owing to their anatomic structure, their function and their environment, are in a position that renders them susceptible to all forms of trauma: chemical, thermal and mechanical. Nature, to compensate for this, has given the oral mucosa the greatest antagonistic power against bacteria of all of the epithelial tissues. The gingival papillae are end-organs, and as such are supplied by end-arterioles. These papillae have a relatively deficient venous back-flow and anything interfering with this function will produce a *locus minoris resistentiae*. It can readily be seen, therefore, that any external trauma, if powerful, or if constantly applied, will pervert the normal activities of the tissue. Likewise, an internal trauma of a toxic nature will produce similar results.

We know that, even in the normal state, the mouth is inhabited by various types of micro-organisms, both pathogenic and nonpathogenic. We know also that in order for the mucous membranes to be attacked, there must be present an organism of high virulence, or a tissue with weakened resistive powers. Infection is an index to us that the normal balance between tissue resistance and bacterial pathogenicity has been upset. Miller, in speaking of Vincent's organisms, said: "The organisms may normally be innocuous, symbiotic saprophytes, capable, under special environment, of causing pathologic changes, possibly under conditions requiring both ancillary organisms and a local damage."

The etiologic factors in the production of Vincent's infection can be classified into two groups: (1) local or direct irritation from within the oral cavity itself; (2) remote or indirect irritation from some tissue or organ of the body which predisposes the oral mucous membrane to infection by lowering its resistance.

*This is the second in a series of articles by Dr. Daley on Vincent's infection.

In studying cases of Vincent's infection, we find, as a rule, that it is not difficult to ascertain the conditions which are causing a local irritation. To locate the remote factors, even though they are seemingly quite evident, it is best to enlist the aid of a thoroughly competent physician.

We believe that Vincent's infection, in most instances, is the end-result of improper hygiene of the oral cavity. This condition may be caused by willful neglect, or may be due to the inability of the individual to cope with a condition arising from a remote etiologic factor.

I shall discuss the local etiologic factors in what seems to me their relative importance. Most of the positive cases that we see show complete or partial neglect of the fundamentals of oral hygiene. Particularly is this true in regard to calcific deposits, both salivary and serumal. Such deposits, even though not glaringly evident, will, by mechanical pressure alone, predispose the tissues to bacterial influences. The normal circulation is interfered with to such an extent that the tissues become toxic, and soon we have a condition of stasis to contend with. Ill-fitting crowns and improper restorative appliances, both fixed and movable, will impinge on pericemental attachments and offer retention spaces for the lodgment of débris. Partially erupted teeth having a flap of tissue over them must be considered. This is a common condition in the third molar region. We have noted, in some cases, that a pocket may occur directly behind a fully erupted upper third molar. Broken down roots and fillings with improperly finished cervical margins are responsible for irritation. Malocclusion of a traumatic nature is a factor. Smoking has often been mentioned as a direct factor, but from our experience, unless it is excessive it seems to have but little to do with the disease. Certain individuals have occupations which make it necessary for them to put foreign bodies in their mouths, thus lowering the resistance mechanically. Traffic policemen furnish us with an example of this type.

In considering the remote factors predisposing to Vincent's infection, we are especially concerned with those diseases which tend to be chronic or obscure. They prepare a favorable habitat for the organisms by weakening the oral mucous membranes through toxic products carried by the blood stream. The tissue resistance-bacterial balance is upset so that the organisms do not meet the usual antagonism. Some of the more prominent diseases which come under this classification are tuberculosis, syphilis, diabetes, various types of nephritis, various fevers and febrile conditions. Pregnancy is a factor which should not be overlooked. Occupational diseases, especially metallic poisoning, must be considered. Acidosis, particularly of the digestive tract, may be a factor. Constipation is probably one of the most important conditions contributing to a lowered resistance.

Vincent's infection may be a secondary invader in practically any form of mouth infection. It may run its course concurrent with some other mouth condition. As for the interaction of diseases, Duke and Divily say: "In persons having several infections, each infection may increase the severity of the others whether due to the same or different organisms. Chronic infections of widely varying types may have a deleterious influence on oral sepsis and render it difficult to cure and prone to recur, and oral sepsis may increase the severity of other chronic infections localized in other organs,

whether due to the same or totally different organisms."

It has been our experience that once the denture has been involved by Vincent's infection, it is never quite up to its previous normal. This disease, unlike many others, does not seem to endow the individual with an immunity to future attacks. The ideal of dentistry today is prevention, and if by careful examination and treatment, we are able to eradicate all, or a few of these predisposing factors, we shall be doing a worthwhile service.

343 Market Street.

# RECOGNITION OF BACILLUS ACIDOPHILUS ASSOCIATED WITH DENTAL CARIES: A PRELIMINARY REPORT*

By FAITH P. HADLEY, M.S.; R. W. BUNTING, D.D.Sc., and EDNA A. DELVES, M.S., Ann Arbor, Mich.

THE recognition of *B. acidophilus* has, in recent years, become a problem of special interest to both the dentist and the bacteriologist. The interest of the dental profession has been awakened by the many reports dealing with the etiologic relationship of this organism to dental caries, and by the hope that a practical method for the recognition and control of this infection might be disclosed. This widespread interest of the dental profession has, in turn, stimulated many bacteriologists to renew and extend the earlier work on the cultural manifestations of *B. acidophilus*. In addition, those bacteriologists interested in *B. acidophilus* as an agent for the relief of putrefactive intestinal disorders have attempted to prove that the organism present in dental caries is not *B. acidophilus*. In this manner, they hope to avert any criticism involved in the suggestion that the frequent ingestion of acidophilus milk for the purpose of establishing the organism in the intestinal tract might initiate dental caries. There are many, on the other hand, who believe that these oral aciduric organisms belong to the *B. acidophilus* group. Thus, a controversy has arisen which has a dental as well as a bacteriologic significance.

Reports have been made by several workers in recent years on the characteristics of both intestinal and oral aciduric organisms. Among these, it is sufficient to mention those of Logan,[1] Cruickshank and Berry,[2] Kendall and Haner,[3] Kulp and Rettger [4] and Roos[5] dealing with the

*Work aided by a grant from the American Dental Association.

*Read before the Section on Histology, Physiology, Bacteriology and Chemistry (Research) at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 23, 1930.

1. Logan, W. R.: Pathol. & Bacteriol., 18:527, 1913-1914.

2. Cruickshank, R., and Berry, D. W.: Brit. M. J., 2:944 (Nov. 22) 1924.

3. Kendall, A. I., and Haner, R. C.: J. Infec. Dis., 35:89 (July) 1924.

4. Kulp, W. L., and Rettger, L. F.: J. Bacteriol., 9:357 (July) 1924.

5. Roos, C.: J. Lab. & Clin. Med., 12:1053 (Aug.) 1927.

intestinal types, and those of Kligler,[6] Howe and Hatch,[7] McIntosh, James and Lazarus-Barlow,[8] Rodriguez[9] and Bunting and Palmerlee[10] on the oral types. More recently, Morishita,[11] working with Rettger, has made a comparison of oral and intestinal strains and has concluded, on the basis of certain cultural differences between the strains, that "the

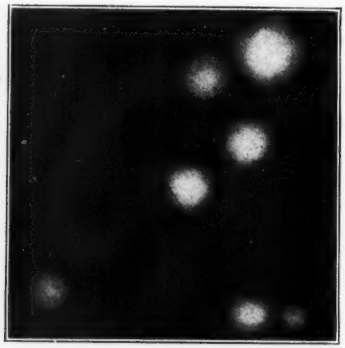

Fig. 1.—Smooth colonies of oral B. acidophilus, Group I, strain 106; 2-day casein-digest agar. (×13.)

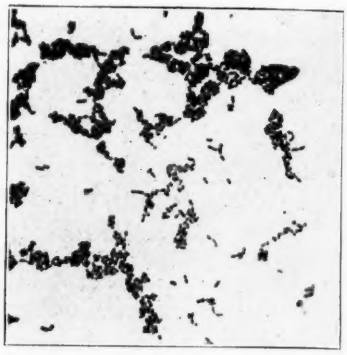

Fig. 2.—Cells from colonies shown in Figure 1. (×1,000.)

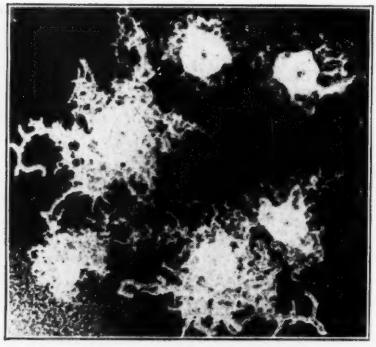

Fig. 3.—Rough colonies obtained from smooth colonies shown in Figure 1; strain 106; 2-day glucose infusion agar. (×13.)

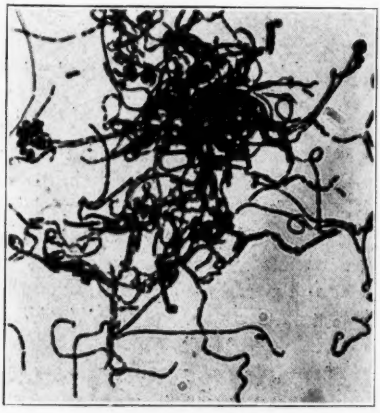

Fig. 4.—Cells from colonies shown in Figure 3. (×1,000.)

6. Kligler, I. J.: J. Allied Dent. Soc., 10:141, 282, 445, 1915.

7. Howe, P. R., and Hatch, Ruth E.: Dent. Cosmos., 59:961 (Oct.) 1917.

8. McIntosh, James; James, W. W., and Lazarus-Barlow, P.: Brit. Dent. J., 43:728 (Aug. 1) 1922.

9. Rodriguez, F. E.: Mil. Dent. J., 5:199, 1922.

10. Bunting, R. W., and Palmerlee, Faith: Rôle of Bacillus Acidophilus in Dental Caries, J.A.D.A., 12:381 (April) 1925.

11. Morishita, T.: Proc. Soc. Exper. Biol. & Med., 25:654, 1928.

tooth organisms constitute a type entirely separate from *B. acidophilus* of the intestinal tract." A later publication by

Fig. 5.—Ropelike filaments from 18-hour glucose broth culture of rough colonies, strain 106. (×1,000.)

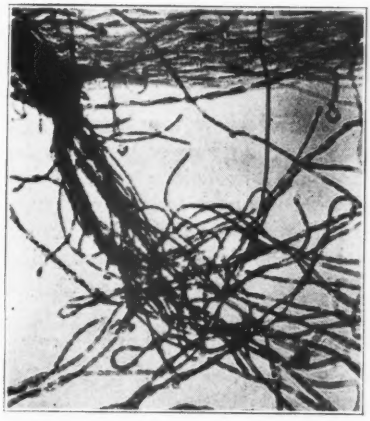

Fig. 6.—End of rope shown in Figure 5.

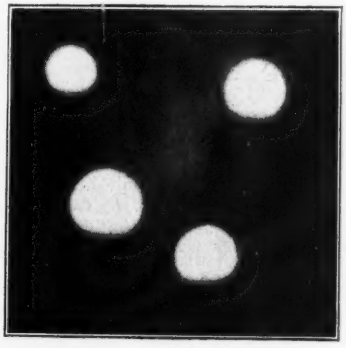

Fig. 7.—Smooth colonies of oral B. acidophilus, Group II, strain 100; 3-day casein-digest agar. (×13.)

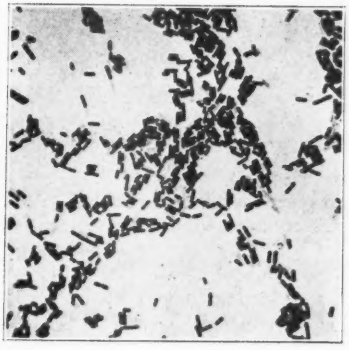

Fig. 8.—Cells from colonies shown in Figure 7. (×1,000.)

the same author[12] states that "it is impossible to say definitely whether the different organisms described by various workers are entirely distinct species, or whether they are different forms of one or the same species." After publication of Morishita's reports, Rosebury, Linton and Buchbinder,[13] in 1929, and Howitt,[14] in 1930, reported results of comparative

12. Morishita, T.: J. Bacteriol., 18:181 (Sept.) 1929.

13. Rosebury, T.; Linton, R. W., and Buchbinder, L.: J. Bacteriol., 18:395 (Dec.) 1929.

14. Howitt, Beatrice: J. Infect. Dis., 46:351 (May) 1930.

studies of organisms from the two sources, and concluded that the cultural differences existing between oral and intestinal strains also existed between the intestinal strains as a group, and that there was therefore no justification in separating these organisms into distinct groups.

All of the above mentioned workers have agreed that both oral and intestinal strains are variable in certain characteristics. Some, especially those working on the oral group, have based their conclusions on the shape of the cells alone. Thus, Howe and Hatch named certain curved aciduric rods, "bacillus M"; others appearing in long chains or filaments, they called "bacillus X"; certain granular filaments they named "bacillus Y"; and short, straight rods they called *B. acidophilus*. McIntosh and his collaborators divided oral aciduric rods into two types: *B. acidophilus-odontolyticus*, Types I and II, on the basis of cell structure and the fermentation of sucrose. Rodriguez separated his strains into three morphologic types, and Morishita[12] described four; while recently Howitt has classified her strains into nine types on the basis of fermentation of eight carbohydrates.

Colony variability has been mentioned, especially for intestinal strains, since Rettger and Horton,[15] in 1914, described their two types, X and Y. The X type, in deep agar, was irregular in shape, with a fuzzy, filamentous border, while the Y type was oval or seed-shaped, with an even outline. Kulp and Rettger, and Morishita, believe the X type to be the usual one recovered from the intestinal tract, while Roos believes that the Y type is found more frequently, although he states that milk drinking favors the development in the intestines of the X type. Morishita reported only the Y type recovered from the mouth, while McIntosh and collaborators, Bunting and Palmerlee, Rosebury and collaborators, and Howitt have all encountered both types.

Since it is the general opinion, therefore, that *B. acidophilus* has different cell forms, different colonies and variable fermentative characteristics, how can we

Fig. 9.—Rough colonies (Variant I) obtained from smooth shown in Figure 7; strain 100; 10-day casein-digest agar. (×13.)

Fig. 10.—Beaded and solid filaments from colonies shown in Figure 9. (×1,000.)

15. Rettger, L. F., and Horton, G. D.: Centralbl. f. Bakteriol., 73:362, 1914.

determine with certainty its presence or absence in the oral cavity, which is swarming with other bacterial forms of all shapes, sizes and cultural characteristics? The methods employed heretofore for detecting the presence of *B. acidophilus* in the mouth have undoubtedly been reliable in the majority of cases, but it is desirable to make these tests, if possible, still more reliable as an indicator of potential dental caries. Our work in this line has also been hindered to some extent by the lack of a fairly simple procedure for ascertaining how many organisms are present. The development of such a quantitative test also depends on the ability to recognize the infecting organism in all of its forms. The workers mentioned above have noted that variations exist, but their observations have been more or less random notations on the appearance of many strains at different times. No thorough or systematic study has as yet been made of the complete range of variability of one strain of *B. acidophilus*, and of the factors which influence that variability. The object of the present investigation has been, therefore, to ascertain what morphologic and cultural variants can be obtained under laboratory conditions from oral and intestinal strains of *B. acidophilus;* and then to compare these variants with similar forms in the mouth, forms which have never been named or classified, and which might never be linked with the *B. acidophilus* group unless studied from this standpoint.

Fig. 11.—Beaded filaments and saclike structures from 17-hour glucose broth culture of rough colonies, strain 100. (×1,000.)

Fig. 12.—Beaded filaments and saclike structures from 17-hour glucose broth culture of rough colonies, strain 100. (×1,000.)

## METHODS

In initiating this investigation, it was necessary to accept some obvious, though perhaps arbitrary, basis for the grouping of the cultures studied. We therefore took for this basis, as earlier invesigators have often done, the morphology of the cells (straight rod, curved rod, cocco-

bacillus, filament, etc.) as they appeared in mouth cultures grown in glucose infusion broth, $p_H$ 5.0. In these cases, observations were also recorded dealing with the appearance of the colonies on agar, as to whether large or small, round or irregular, smooth or rough.

When tentative groupings on the basis of cell structure had thus been established, it would be feasible to attempt correlations with other important characters, such as the fermentation of sugars, acidogenic properties and serologic reactions; and, in addition, to ascertain whether any of the group characters were interconvertible, or showed further variations. It was also necessary to keep in mind the morphologic groupings that have been outlined by earlier workers, and to relate, if possible, the types described by one worker to those of another. The lack of illustrations in the majority of articles on oral strains, and the meager descriptions offered by some workers, have made this a difficult task.

The oral strains employed in this study were secured by our usual method, direct inoculation of scrapings from carious teeth into tubes of 1 per cent glucose infusion broth, $p_H$ 5.0. After incubation at 37 C. for from two to four days, cultures were plated on casein-digest sodium oleate-agar. Surface spreadings were made use of almost entirely. Colony types were noted, and each particular type replated two or three times to insure purity and stability. One per cent glucose infusion agar, $p_H$ 7.0, was often used after the original platings, in order to study more carefully the colony structure, which is made difficult by the opacity of the casein-digest agar. Stock strains were carried on casein-digest agar slants.

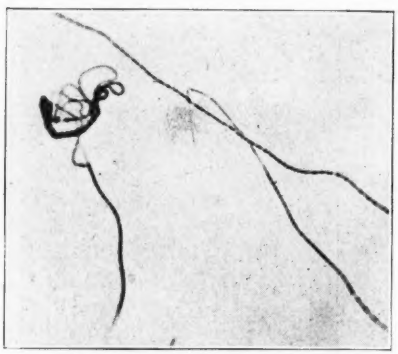

Fig. 13.—Beaded filaments and saclike structures from 17-hour glucose broth culture of rough colonies, strain 100. (×1,000.)

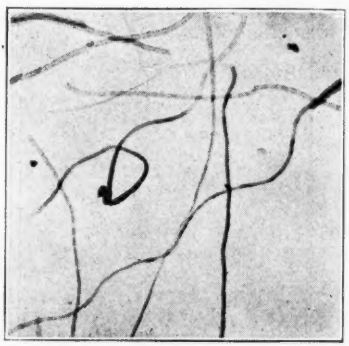

Fig. 14.—Beaded filaments and saclike structures from 17-hour glucose broth culture of rough colonies, strain 100. (×1,000.)

The intestinal strains studied at this time were as follows: 833: American type culture collection; original from Rettger (Rettger "scar"); 314: American type culture collection; original from Rettger (Rettger 43), and 110: from National Antitoxin and Vaccine Institute, Washington, D. C.; isolated from intestinal tract and used in commercial preparation of acidophilus milk.

CELL MORPHOLOGY AND COLONY CHARACTERISTICS OF ORAL STRAINS

The following groups are presented in a tentative scheme for classification of these oral aciduric rods. They represent the morphologic types most frequently recovered from carious lesions.

*Group I.*—These small, gram-positive coccobacilli usually appear in chains in broth, and in pairs on agar. The palisade arrangement is not marked. The rods are often curved, with concave sides approximate. Two or three adjoining organisms may form a circle. The variation in size is very slight. (Fig. 2.)

Colony: The surface colony on casein-digest agar in two days is about 0.8 mm. in diameter, round, smooth, glistening and opaque. It varies from slightly flat to moderately convex, and is very stable. (Fig. 1.) Deep colonies are seed-shaped, resembling Rettger's Y type.

This organism resembles in structure the bacillus M of Howe and Hatch. It may be identical with *Lactobacillus odontolyticus,* Type II, of McIntosh and collaborators, and Type II of Rodriguez. It also resembles in structure and when in colony Group I described by Morishita.

*Group II.*—These bacilli are also gram positive, but slightly longer than those of Group I. There is no tendency toward chaining, but the rods show definite palisade arrangements (both on agar and in broth). Variation in size is slight. Occasional filaments may be seen. (Fig. 8.)

Colony: Two-day surface colonies on casein-digest agar are from 1.0 to 1.2 mm. in diameter, round, smooth, white, glistening and distinctly convex. They are fairly unstable, as will be shown later. Deep colonies are of the Y type. (Figs. 7 and 39.)

This is probably the organism described by Howe and Hatch as *B. acidophilus,* and may be the Type I of McIntosh and collaborators and of Rodriguez. In structure, it resembles the organisms in Group

Fig. 15.—Dark-field photomicrograph of rough culture of strain 100; 48-hour broth culture. (×700.)

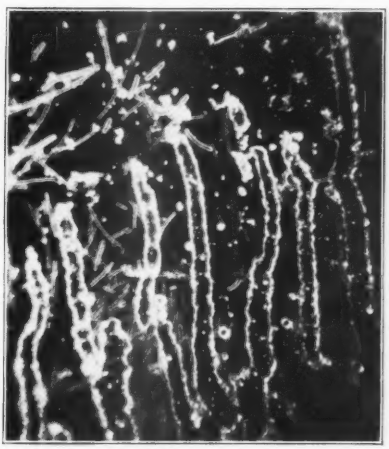

Fig. 16.—Dark-field photomicrograph of rough culture of strain 100; 48-hour broth culture. (×700.)

II of Morishita, although his colony description does not agree with the foregoing.

*Group III.*—This is a thicker, more blunt bacillus than that of Group II, especially on agar. In broth, it may closely resemble the Group II organisms. The length of cells on agar is variable, some being very short and coccoid, others longer but thick. (Fig. 25.)

Colony: The surface colony on casein-digest agar was about 1.5 mm. in two days. It was characterized by its flat, gray appearance, slightly uneven outline, and smooth surface, and was often rimmed, with a central depression, as shown in Figure 24. The deep colony was fuzzy, being similar to Rettger's X type.

This type may correspond to Morishita's Group III, with which it seems to coincide more closely in cell form than in colony characteristics.

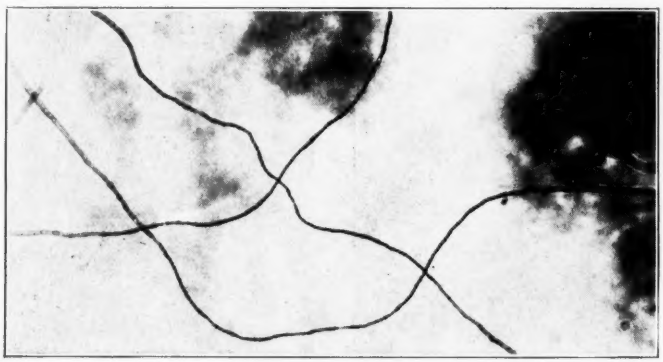

Fig. 17.—Extremely long filaments of rough culture of strain 100; 48-hour litmus milk. (×1,000.)

### BIOLOGIC REACTIONS OF FOREGOING GROUPS

*Acid Production.*—Tests for acid production were carried out in a 2 per cent glucose infusion broth, $p_H$ 6.2, $p_H$ readings by the colorimetric method being taken at two and seven day intervals. It was found that the Group I strains produced an average acidity of $p_H$ 3.8 in seven days, while the strains of Group II and III reached only 4.1.

*Litmus Milk.*—Group I strains produced acid and clotting in litmus milk in from one to three days, while those of Groups II and III clotted milk very slowly, requiring from three to four weeks. This is in accordance with Morishita's observations on his groups.

*Fermentation of Carbohydrates.*—Strains of the three groups were tested on eighteen carbohydrates. Casein-digest broth, $p_H$ 7.0, made from lactose-free casein was employed, to which was added, in sterile aqueous solution, 1 per cent of the carbohydrate. All sugars were sterilized by filtration. Readings were taken in $p_H$ at the end of four and ten days.

Group I: Some strains fermented the pentoses, xylose and rhamnose, others did not. Arabinose was broken down by none. Dextrose, galactose and mannose were rapidly fermented by all. Strains were variable on maltose and sucrose, but all fermented lactose and trehalose. The

trisaccharid, raffinose, was fermented by none, while all produced acid in melezitose. Dextrin was not fermented. Strains were variable on inulin and dulcitol, but all were positive on sorbitol, mannitol and salicin. It is thus seen that nine of the eighteen sugars were broken down by all of the Group I strains.

Group II: All strains of this group fermented only dextrose, galactose, sucrose, lactose and raffinose. The reaction was variable on arabinose, mannose and maltose, while none fermented xylose, rhamnose, trehalose, melezitose, dextrin, inulin, sorbitol, dulcitol, mannitol and salicin.

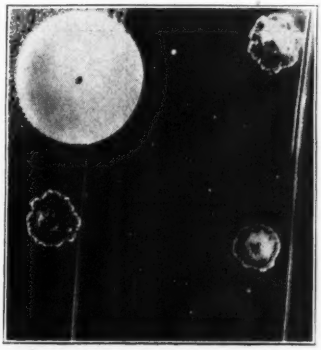

Fig. 18.—Variant II colonies obtained from smooth colonies of strain 100; three Variant II colonies and one smooth may be seen; 3-day glucose infusion agar. (×13.)

Fig. 19.—Cells from Variant II colonies shown in Figure 18. (×1,000.)

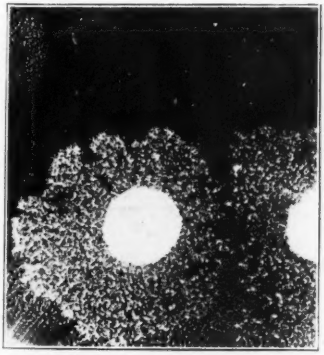

Fig. 20.—Variant III colonies obtained from rough (Variant I) of strain 100; 7-day glucose infusion agar. (×9.)

Fig. 21.—Granular rods from Variant III colonies shown in Figure 20. (×1,000.)

Group III: These strains were even less active than those of Group II. They fermented dextrose; they were variable on arabinose, galactose, mannose, mal-

tose, sucrose, lactose, trehalose and raffinose, and all were negative on xylose, rhamnose, melezitose, dextrin, inulin, sorbitol, dulcitol, mannitol and salicin.

*Esculin Fermentation.*—The splitting of esculin has been used for some time as a differential test for the colon bacillus.

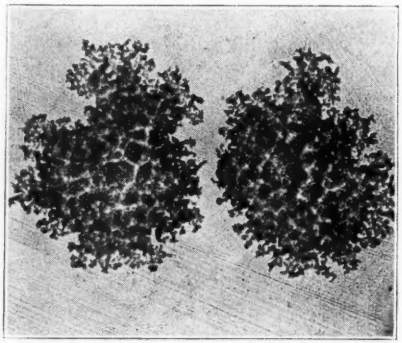

Fig. 22.—Variant IV colonies obtained from Variant I or Variant III of strain 100; 3-day glucose infusion agar. (×13.)

Fig. 23.—Cells from Variant IV colonies shown in Figure 22. (×1,000.)

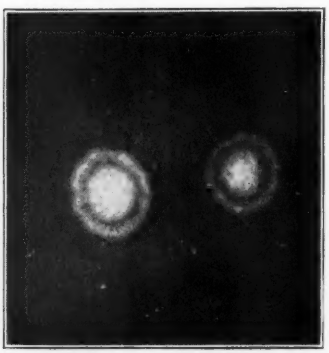

Fig. 24.—Intermediate colonies of oral B. acidophilus, Group III, strain 103; 3-day casein-digest agar. (×13.)

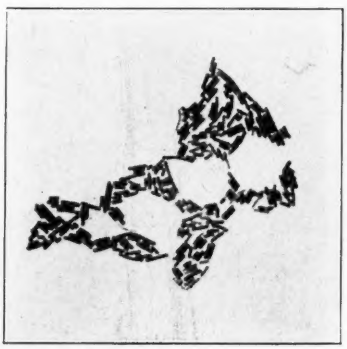

Fig. 25.—Cells from colonies shown in Figure 24. (×1,000.)

More recently, it has been employed as one of the tests for distinguishing the enterococcus (*Streptococcus fecalis*) from the viridans streptococci. Since the fermentation of mannite and salicin, and the lack of fermentative ability for raffinose, is characteristic of the enterococcus, and since this was characteristic of all strains of the foregoing Group I of oral *B. acidophilus,* the esculin test was applied to strains of the aciduric organisms.

Esculin bile broth, as recommended by Meyer and Schönfeld,[16] was used for the

16. Meyer, K., and Schönfeld, H.: Centralbl. f. Bakteriol., 99:402, 1926.

tests. It was found that all strains of Group I were positive on this medium, while all strains of Groups II and III were negative. There seems to be a correlation, therefore, both in the enterococcus group and in the Group I of oral

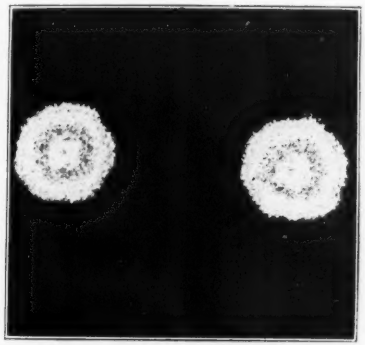

Fig. 26.—Rough colonies obtained from intermediate colonies shown in Figure 24; strain 103; 2-day glucose infusion agar. (×13.)

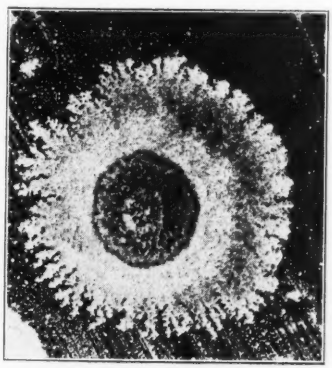

Fig. 28.—Older rough colony of strain 103; 12-day glucose infusion agar. (×9.)

*B. acidophilus,* between the ability to break down mannite and salicin and to ferment esculin.

DISSOCIATION OF B. ACIDOPHILUS

The three groups of aciduric organisms described above represent the forms most commonly recognized in cultures

Fig. 27.—Cells from rough colonies shown in Figure 26; branching form. (×1,000).

Fig. 29.—Cells from colony shown in Figure 28. (×1,000.)

from caries. All of the three groups appear to have certain characteristics in common, as well as peculiarities which are distinct enough to justify their sepa-

ration into three groups. What would be the result if a representative strain from each group were broken up into all possible variants? Would the variants from one group be identical with those of another, and, through these variants, could a Group I strain be converted into a Group II or III strain? Could the variants secured under artificial conditions be related to organisms in the mouth which have never been linked with *B. acidophilus?* Would these variants possess aciduric and acidogenic properties, or any other properties, which might be important as factors in the production of dental caries? Also, what relation would these variants bear to the intestinal types of *B. acidophilus?*

This problem was attacked by attempting to dissociate a typical strain of each of the three groups of oral aciduric organisms. The more detailed methods will be presented elsewhere. Briefly stated, this attempt involved a careful study of colony variation in pure-line strains, with the cultivation from the rough, filamentous borders or marginal outgrowths which occur under certain conditions on the common, smooth type of colony. Figures 36 and 37 illustrate dissociating colonies of the Group I and II strains, from which the "rough" variants were derived.

*Group I Dissociation.*—Figure 1 shows the "normal" type of smooth (S-type) colony as isolated from the mouth. Figure 2 shows the short, coccobacillus from this colony. When an S-type colony is inoculated into glucose broth, the clouding is homogeneous, with little, if any, precipitate. Figure 3 shows the larger, spreading "rough" (R-type) colony derived from the smooth; and Figure 4, the chains of rods and filaments contained in this rough colony.

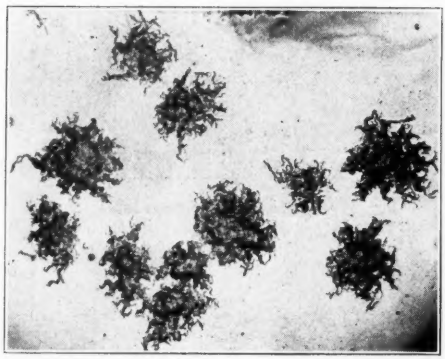

Fig. 30.—Rough colonies of intestinal B. acidophilus, strain 833 (Rettger "scar"); 3-day glucose infusion agar. (×13.)

Fig. 31.—Cells from colonies shown in Figure 30. (×1,000.)

When this rough colony is inoculated into glucose broth, growth occurs in from twenty-four to forty-eight hours in the form of a granular precipitate, the clumps of which are so coherent that only the most vigorous shaking can break them up. The supernatant broth is entirely clear. When these clumps are examined microscopically, the reason for their coherence is easily seen, for they are com-

posed of long filaments united in tangled skeins or braided ropes. In places, the filaments are broken up into long chains of cocci or rods. The end of one of these ropes may be seen in Figures 5 and 6. The entire rope extended across several microscopic fields.

These filaments, if seen in direct smears from the mouth, would undoubtedly be called leptothrix. They are seen to form only a phase (but possibly the most important phase) of the life history of this group of oral aciduric rods. These filaments can grow in, and produce, as much acid as the smooth culture from which they come. Clinically, they are doubtless far more important than the smooth type of culture, because of their spreading, sticky and coherent form of growth, which would facilitate their attachment to the tooth surfaces. Plaque formation is undoubtedly aided by bacterial forms such as these, and because of this fact, coupled with their high acid-producing power, they become a factor of considerable interest and importance in the bacteriology of caries.

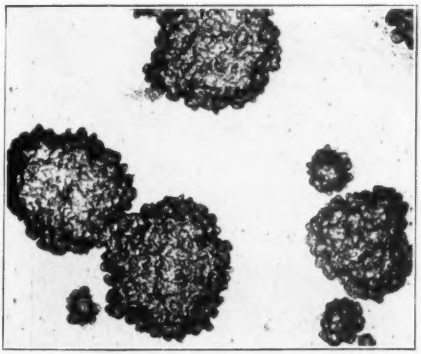

Fig. 32.—Rough colonies of intestinal B. acidophilus, strain 314 (Rettger 43); 3-day casein-digest agar. (×13.)

Fig. 33.—Cells from colonies shown in Figure 32. (×1,000.)

Although the S-type of this strain clotted milk rapidly, the R-type acted more slowly. The fermentation of certain sugars also differed. Whereas, the S-type culture was negative on maltose and positive on sorbitol and mannite, the R-type was positive on maltose but negative on sorbitol and mannite. Both S and R fermented esculin.

*Group II Dissociation.*—Figures 7 and 8 show the colony and cell form of the "smooth" type of culture of a Group II strain. The growth in broth is homogeneous. Colonies frequently show marginal dissociation, and Figure 9 illustrates the "rough" type of colony secured in this manner from the smooth. This R culture, like the R of Group I, is made up of filaments, both solidly stained and granular or beaded (Fig. 10). In broth, the growth occurs as a soft, flocculent precipitate in the bottom of the tube. Coiled and braided ropes (of filaments), which were described above for the R culture of the Group I strain, have not been observed. Very long and tangled filaments were seen; and, in addition, sac-like structures attached to certain

filaments, were often observed. Stained specimens of these are shown in Figures 11-14. It should be noted that these appeared in very young broth cultures (17-18 hours), and, therefore, these structures cannot be interpreted as "involution forms." In fact, they disappeared entirely from older cultures.

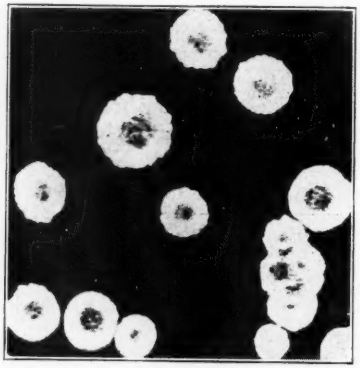

Fig. 34.—Smooth colonies of intestinal B. acidophilus, strain 314; 3-day casein-digest agar. (×13.)

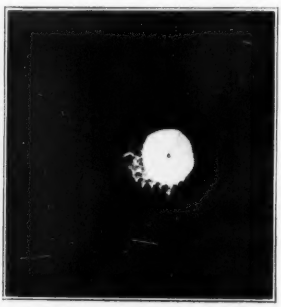

Fig. 36.—Smooth colony of oral strain 106, showing marginal dissociation to rough colony; 2-day glucose infusion agar. (×13.)

The nature and significance of these structures cannot be explained at the present time. They are not unlike some of the forms of *Leptothrix racemosa* described in 1897 by Vicentini[17] and in 1899 by J. Leon Williams.[18] The beaded filaments were especially emphasized by Vicentini as being a part of the cycle of development of the "fructification ears" of his *Leptothrix racemosa.* Certain dark-field preparations of these broth cultures of the R type of *B. acido-*

Fig. 35.—Cells from colonies shown in Figure 34. (×1,000.)

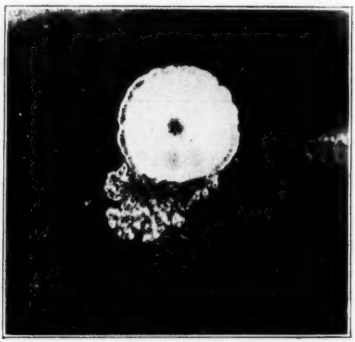

Fig. 37.—Smooth colony of oral strain 100 showing marginal dissociation to rough colony; 9-day casein-digest agar. (×13.)

17. Vicentini, F.: Bacteria of the Sputa and Cryptogamic Flora of the Mouth, 1897.

18. Williams, J. L.: Dent. Cosmos., 41:317 (April) 1899.

*philus* reveal stalklike elements, both intact and partially broken down, which distinctly remind one of certain photographs of *L. racemosa* published by J. Leon Williams, and more recently by Beust.[19] (Figs. 15 and 16.) Since these investigators did not cultivate this leptothrix on artificial culture mediums, but found it necessary to confine their observations to direct microscopic preparations from the mouth, it is difficult to make any direct comparisons between the two forms.

Another variant (Variant II) which it has been possible to obtain from the

Fig. 38.—Deep colonies of rough intestinal strain 833, showing the Rettger X-type of colony; 4-day whey agar. (×13.)

"smooth" culture of this Group II strain is shown in Figure 18 (one smooth colony and three of this second variant are shown). This variant was smaller than the smooth colony, flat and slightly irregular in outline. The cells composing this type of colony are shown in Figure 19. The wavy, curved and hooked appearance was characteristic. These colonies were very unstable, soon reverting to the S-type, especially in broth.

Variant III (Fig. 20) is a type of culture which arose from the R culture (shown in Figure 9). When this colony was 24 hours old, it appeared white, convex and smooth, and one might believe that the R culture was reverting directly to the S. By the time it was 48 hours

Fig. 39.—Deep colony of smooth oral strain 100, showing the Rettger Y-type of colony; 4-day whey agar. (×13.)

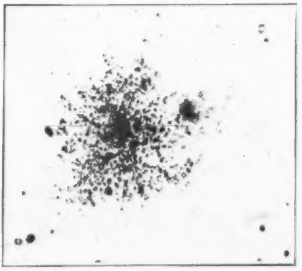

Fig. 40.—Deep colony of rough oral strain 100, showing the Rettger X-type of colony; 4-day whey agar (compare with Figure 38). (×13.)

old, a thin, filamentous halo had begun to develop, which continued to extend for several days. The central portion of the colony, instead of being opaque like the S colony, had a definite cutglass appearance. The cells from this type of colony,

19. Beust, T. B.: J.A.D.A., Morphology and Growth of Fusiform Organisms, 16:714 (April) 1929.

shown in Figure 21, were always granular, the granules usually lying along both sides of the rod, just within the cell wall, in a very characteristic manner, giving the rods a "moth-eaten" appearance.

Variant IV (Fig. 22) is a very thin, spreading, lacy colony, arising both from the R culture (Variant I) and from Variant III. Some colonies of Variant III always appeared after passage of Variant IV through broth. This colony is characterized by cells highly pleomorphic, great variation being shown not only in length but also in width (Fig. 23). Comma forms, hooks, loops, spirochete-like forms, and filaments could all be seen in a single preparation from a single young colony.

The fermentation reactions of the foregoing variants differed only slightly from those of the smooth mother type. Variant IV fermented mannose, which the others did not.

### AGGLUTINATION

Rabbit immune serums were prepared against the S-type of the foregoing Group II strain (Strain 100), and against the R-type (Variant I) of the same strain. The S antigen was completely agglutinated in its own serum in a dilution of 1:2,560, while, in the R serum, it was completely agglutinated through a dilution of 1:1,280. The R antigen was completely agglutinated in the R serum through 1:10,240, and in the S serum through 1:5,120.

*Group III Dissociation.*—Figures 24 and 25 show the typical colony and cell form of a Group III strain. This type was not a true "smooth" colony, but rather an intermediate between smooth and rough. On infusion agar, the colonies resembled those of Variant II of the Group II strains. It was possible to dissociate it into a true "rough" which greatly resembled the rough Variant IV of the Group II strain (compare Figures 28 and 29 with 22 and 23). The colony was thin, lacy and transparent, and contained the same curved, hooked and comma forms of cells.

### DISSOCIATION OF INTESTINAL STRAINS

*Strain 833.*—This strain, originally from Rettger, was in the "rough" stage, corresponding to the rough variants described above from smooth oral strains. Figure 30 shows these rough, filamentous surface colonies of strain 833, and Figure 31 the coiled, looped, irregularly shaped organisms which make up these colonies. Figure 38 shows the deep colonies of this strain, colonies which Rettger has designated as "X" (compare with Figure 40, which shows a deep colony of the "rough" variant of our oral *B. acidophilus* 100).

*Strain 314.*—This, which was also from Rettger's laboratory, was also typically rough in character, as may be seen in Figure 32. The cells from these colonies varied greatly in length, width and staining ability (Fig. 33), many having the granular appearance described for certain stages of the oral strains. (Compare with Figures 9 and 10.) We were able to dissociate this rough strain into a type which gave a fairly smooth colony. In this phase, the cells were shorter, straighter, more uniform in size, and more comparable to those in the usual smooth form of the oral acidophilus (Figs. 34 and 35).

*Strain 110.*—This, an intestinal strain used for the commercial manufacture of acidophilus milk, was a smooth type of colony, resembling those of our oral Group I, and showing small coccobacilli in chains. The rods were often curved,

as described above for our Group I oral strains.

The fermentation characteristics of the three foregoing strains were variable. Strain 110, a "smooth" type, closely coincided with the reactions of our oral Group I, in fermenting dextrose, galactose, mannose, lactose, trehalose, sorbitol, mannitol and salicin. It also fermented esculin. The Rettger strains, although fermenting esculin, did not ferment melezitose, sorbitol or mannitol, but did break down trehalose, raffinose and salicin. They therefore possessed some fermentative characteristics of all three oral groups.

## SUMMARY AND CONCLUSIONS

The characteristics of three groups of oral aciduric organisms from dental caries have been described and compared with intestinal strains. The first group ("smooth") had greater acid-producing power than the other two groups ("smooth" and "intermediate"), and was capable of breaking down into acid a larger number of carbohydrates than the others. It was a sorbitol-mannitol-salicin fermenter and a raffinose nonfermenter—characteristics which Morishita mentioned for his Group I strains. It also fermented esculin. The other two groups were sorbitol-mannitol-salicin-esculin nonfermenters, but, as a rule, raffinose-fermenting.

Of three intestinal strains studied, one ("smooth") showed all the characteristics of the oral Group I. The other two ("rough") were sorbitol-mannitol negative, but raffinose-salicin-esculin positive.

A representative strain from each of the oral groups was dissociated into pure "rough" strains which had many of the morphologic and cultural features of the "rough" intestinal strains. A "rough" surface colony was shown to correspond to Rettger and Horton's deep "X" type, while the "smooth" colonies showed the characteristics of their "Y" type. Since, by simple laboratory procedures, the smooth oral strains (which are usually recovered from the mouth in this phase) can be transformed into the "rough," and since rough intestinal strains (which are often recovered from the intestines in this stage), can be changed into the "smooth," we contest the published statements of Morishita and Rettger that the oral strains can be differentiated from the intestinal on the basis of colony appearance.

On the same grounds, we disagree with their conclusion that the intestinal *B. acidophilus* is characterized by growth in long chains or threads, while the oral strains are characterized by growth in small, short rods. As a matter of fact, the smooth phase of the rough intestinal strains shows short rods, and the rough phase of the smooth oral strains reveals long chains and threads. In other words, both the intestinal strains and the oral strains of *B. acidophilus* occur in two phases, the S and the R; and, for purposes of revealing species differences, it is scarcely logical to compare the R-phase of one culture with the S of another.

That this has actually been done is shown by a recent article by Hunt and Rettger.[20] These authors state, "Morishita has noted that the organisms isolated by himself and others from carious teeth are, with few exceptions, not *L. acidophilus as this species or type has been described by Kulp and Rettger* (italics ours) and by others." This statement undoubtedly shows why Rettger and his associates have been unable to recognize

20. Hunt, G. A., and Rettger, L. F.: J. Bacteriol., 20:61, 1930.

the oral acidophilus strains as belonging to the *B. acidophilus* group. Their own descriptions of this species have always been based on a study of the "rough" or "X" type of culture, which they consider the "typical" *B. acidophilus*. They have not given consideration to the smooth type. Such a point of view clearly implies either a disregard for, or ignorance of, the important phases in the life history of a bacterial species, with which they should be in a position to claim considerable familiarity.

The long, leptothrix-like filaments which make up the "rough" types of *B. acidophilus* are interesting from a clinical point of view in that they may be closely concerned in the process of plaque formation on the tooth. This phase of the problem is being studied in greater detail.

# A SURVEY OF CURRENT INLAY CASTING TECHNICS*

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ONE of the items which have been incorporated into the cooperative research program of the American Dental Association and the National Bureau of Standards is a survey of inlay casting technics. The dentist has been more or less confused by the number, diversity and conflicting details of methods and devices offered to him since the advent of the cast gold inlay. This survey was undertaken with the idea of obtaining a comparison of the various processes made by unbiased observers with no commercial affiliations.

Thousands of dollars have been spent and are being spent by the dental profession on special clinics and lecture courses which are presented by clinicians and lecturers advocating empiric and arbitrary methods without sound scientific foundation, not always because they are interested in progress, but often because they are interested primarily in selling special materials, equipment or service.

We hope to present in this paper a sufficient number of data on the fundamentals of the casting process to enable the individual dentist to analyze critically any new process submitted to him. If he will take the time necessary to do this before signing on the dotted line, he will be in a position to make it exceedingly embarrassing and uncomfortable for the next high pressure salesman who tries to sell him Dr. Blank's *special* inlay furnace,

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*special* casting machine or *special* investment.

Comparison of current inlay casting technics was made with the view of determining the extent to which the variables of the casting process were controlled. R. L. Coleman[1] has reported that the average shrinkage of dental gold castings is approximately 1.25 per cent. The production of an inlay that actually fits requires that this shrinkage be balanced by producing an expanded mold through a combination of (1) the thermal expansion of the wax pattern, (2) the setting expansion of the investment, and (3) the thermal expansion of the investment.

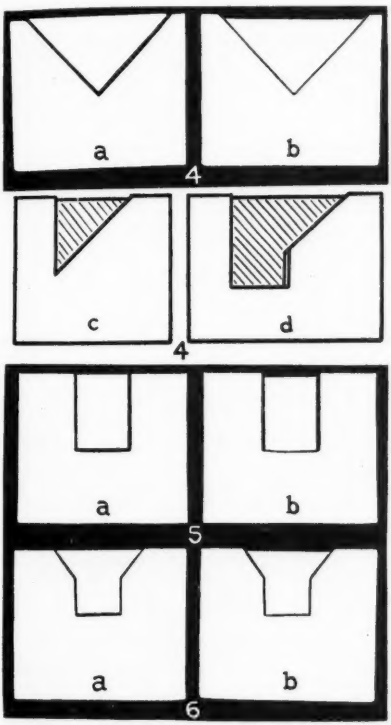


Fig. 1.—*4a, b, c* and *d*, diagrams of cone-shaped cavities where the contraction of the gold casting would not prevent the casting going to place. *5a* and *b*, same as *4* but for cylindrical cavities. *6a* and *b*, same as *4* and *5* but for a combination of conical and cylindrical types of cavity (Solbrig).

The first and third of the three items may be easily determined over the ordinary working ranges, but at present there are few data as to the exact proportion of the setting changes in investments which may serve to compensate for the shrinkage of gold.

Castings were made according to the details of each of the technics studied. For the sake of uniformity and to insure accuracy in observation wax patterns were made of mesioclusodistal cavities prepared in steel dies of the type designed by Dr. Weinstein. We realize that the steel die has been criticized as a "trick cavity" and its use as a "casting stunt" decried. We feel that it presents a most rigid and exact cavity for test purposes as it shows (1) minute amounts of shrinkage; (2) traces of overexpansion, and (3) effects of surface irregularities.

To fit the steel die, a casting must be accurate in dimensions and must have smooth surfaces, two of the primary requisites of a good inlay. The cavities are of such shape that they are exceedingly difficult to fit unless the variables in the casting process are carefully controlled.

Oskar Solbrig[2] has presented two diagrams which are of interest as they show the effect of shrinkage of castings when placed in the cavity for which they are

1. Coleman, R. L.: Physical Properties of Dental Materials, Dent. Cosmos, 69:1007 (Oct.) 1927; Bur. Standards J. Res., December, 1928.

2. Solbrig, Oskar: J. Dent. Res., February, 1929, p. 55.

made. These diagrams are reproduced in connection with the discussion of the type of cavity preparation used in the present series of tests, as they show graphically the reasons for the choice of the steel die as a test cavity.

Diagrams *4a, b, c* and *d,* Figure 1, illustrate cone-shaped cavities where the contraction of gold during the casting process will make no apparent variation in fit except for a shortage of gold at the cavity margins. A slight grinding of the enamel would give the appearance of a perfect fit.

Diagrams *5a* and *b* illustrate the fact that in cylindrical cavities, casting shrinkage would be evident both from loose fit and lack of gold at the occlusal surface. This would require the use of generous quantities of cement in addition to the removal of some enamel. Diagram *6a* and *b* shows the combination of cylindrical and taper cavities.

Diagrams *7a* and *b,* Figure 2, illustrate the result of shrinkage in a mesioclusal inlay, while diagram 8 shows the effect of shrinkage in a mesioclusodistal inlay. In both cases, the discrepancies are such that a heavy cement line must result.

Figure 3 is a diagram showing the effect produced when an attempt is made to cast a crown or mesioclusodistal inlay for a tooth having an external taper, without compensating fully for the shrinkage of gold. The taper is a lifesaver in this case, since it prevents the casting from being loose. The use of a generous quantity of cement and of a soft gold which can be burnished to a considerable degree renders it possible to make an apparent but not an actual fit by the various low heat and cold mold and other technics which do not compensate for the shrinkage of gold in casting.

The various technics were carried out exactly as described by certain of their advocates. The data given relative to each technic indicate the details of the process, and when the conditions warrant it, possible remedies are suggested for inaccuracies in the technics. The following technics were reviewed and presented in the following sections in the order named.

1. Low heat method of elimination.[3]
2. High heat method of elimination.[4]
3. Rapid elimination of the wax pattern (Knapp method).[5]

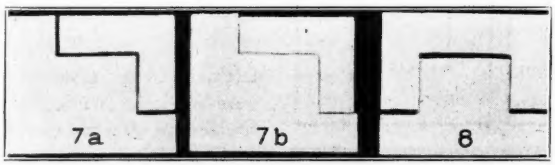


Fig. 2.—*7a* and *b,* illustration of the contraction of a cast mesioclusal inlay, and the resultant space at the gingival seat. *8,* diagram of a mesioclusodistal inlay or full crown where the contraction of the gold proves disastrous (Solbrig).

3. Volland, R. H.: Low Heat Elimination, J. A. D. A., 15:1545 (Aug.) 1928.

4. Grunn, A. P.: High Heat Elimination, J.A.D.A., 15:1535 (Aug.) 1928.

5. Altfillisch, H. J.: Rapid Elimination (Knapp Method), J.A.D.A., 15:1556 (Aug.) 1928.

4. Water bath technics.[6]

5. Meyer-Maves casting technic.[7]

6. The semidirect method of casting inlays.[2]

7. The whip-mix technic.[8]

LOW HEAT ELIMINATION

The low heat elimination technic is essentially as follows:

Fig. 3.—Diagram illustrating fit of a mesioclusodistal inlay or full cast crown on a taper preparation when casting variables are not fully balanced.

6. Van Horn, C. S.: Dent. Cosmos, 52:873 (Aug.) 1910. Rice, W. S.: Dent. Bull., U. S. Army, January, 1929, p. 3. Coe-Furrow Casting Technic, Bull. Coe Laboratories. (This paper and the preceding one are in many paragraphs identical but both are without explanation, reference or credit to the other.) Green Investment Casting Technique, Bull. Ransom and Randolph Co.

7. Maves, H. A.: Control of Variables in the Casting Process, J. A. D. A., 15:1550 (Aug.) 1928. Meyer, F. S.: Some Sources of Error and Their Correction in the Making of Cast Gold Inlays, J. A. D. A., 16:812 (May) 1929.

8. Whip-Mix Technique, Bull. Whip Mix Corporation.

1. A wax pattern adapted to the cavity at near room temperature, free from strains, recently made and removed, is washed with soap suds. 2. A single investment is used which contains at least 50 per cent of plaster of Paris. This is mixed to a creamy consistency and painted on. The remainder is poured around the painted pattern without jarring. 3. All materials are used at room temperature, i.e., the same temperature as the wax pattern. 4. The wax is burned out by heating slowly to 160 C. (320 F.). 5. The mold is allowed to assume room temperature before casting.

Fig. 4.—Equipment used in determining the temperature of wax patterns in the mouth.

The author[3] says, "The wax must be in adaptation with cavity details at a temperature closely approximating that of the room." In contrast to this statement, we find that Dr. Van Horn[9] says that, in considering wax pattern temperatures in the mouth, we should consider mouth temperature or near mouth temperature as the temperature at which the wax will be in adaptation to the cavity details.

An experiment was performed to determine the temperature of wax patterns in the tooth *in situ* under varying conditions.

A cavity 4.8 mm. deep and 5.3 by 6.5 mm. on the occlusal surface was prepared in a lower first molar. A wax pattern

9. Van Horn, C. S.: Expanded Pattern Casting Technic, J. A. D. A., 17:763 (May) 1930.

with a thermocouple embedded in it was made on an indirect amalgam model and transferred to the dry cavity and sealed. (Fig. 4.) It rapidly assumed a temperature near to that of the body, 36.63 C. (98 F., approximately). Room temperature water, 26 C. (78.8 F.), was used to cool the pattern. After the pattern had been bathed for three minutes, the temperature of the pattern had dropped only 4 degrees C. (7.2 F.) to 32.85 C. (91 F.). Tap water which was at 18 C. (64.4 F.) was next used. After eight minutes, the temperature of the wax fell to 30.96 C. (87.8 F.). Ice water at 0 C. (32 F.) had to be resorted to in order to bring the pattern to 24.74 C. (77 F.) or just 2 degrees below room temperature, and under those conditions the patient was far from comfortable. The pattern rapidly assumed mouth temperature after the supply of cooling water was cut off.

This experiment was carried out under the same conditions that prevail in actual dental practice. It was carried out in summer when room temperature was 26 C. (78.8 F.). From it, we can see that it is impractical to get the wax pattern in adaptation to cavity detail at room temperature. What if the experiment had been carried out during the winter months with room temperature at 20 C. (68 F.)?

A thermal expansion curve was run on an investment made up of 50 per cent plaster of Paris and 50 per cent silica, by the use of the equipment described by Hidnert and Sweeney.[10] (Fig. 5.) The investment was heated slowly as directed, to a maximum temperature of 160 C. (320 F.), where it was held for three hours. The greatest expansion was 0.13 per cent at 100 C. (212 F.). At 160 C. (320 F.), the expansion was 0.09 per cent. On cooling to room temperature, the mold shrinks further, until it is 0.11 per cent smaller than its original dimensions.

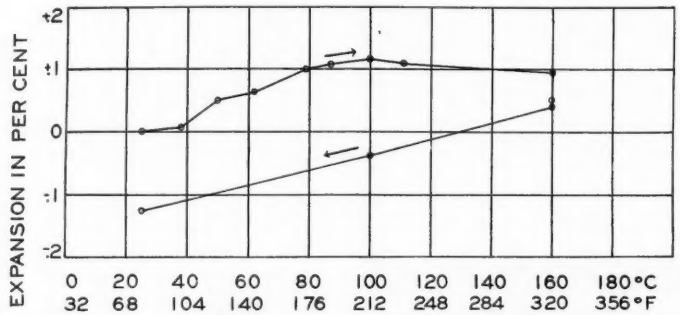


Fig. 5.—Thermal expansion of investment containing 50 per cent silica. Rate of heating: From 25 to 160 C., two hours; at 160 C., two hours. Powder, 100 gm.; water, 60 c.c.

A casting made at 160 C. as directed should compensate for only 0.09 per cent of the 1.25 per cent shrinkage of the gold and be undersize about 1.15 per cent when compared to the cavity. An inlay cast into a mold at room temperature would be about 1.35 per cent smaller than the cavity due to the combined shrinkages of the mold and the gold.

10. Hidnert, Peter, and Sweeney, W. T.: Bur. Standards J. Res., November, 1928 (Figs. 6-7).

It has been suggested by some that the shrinkage of gold from the point of its greatest fluidity to its solidification can be controlled to a certain extent in the casting process. "This is accomplished by bringing the gold to a temperature that is just sufficient to permit it to be forced to place in the mold. Thus, the mold is filled with gold that is expanded to a minimum, and, during the cooling down to solidification, only a minimum contraction will take place."

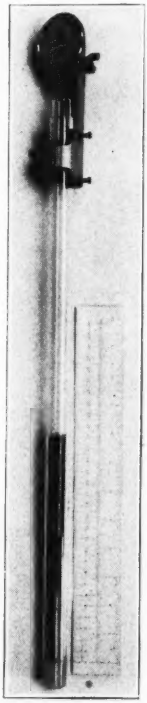

Fig. 6.—Fused quartz tube expansion apparatus.

This does not agree with the findings of Coleman,[1] who reported that gold shrinkage was practically the same when "the temperature of the metal varied from the lowest at which a casting could be made to the maximum temperature obtainable with a good blowpipe using hydrogen and compressed air." This was determined by actual experiment, and, in a practical application, no apparent difference could be observed. Other physical properties of the alloy may be altered by overheating, but not the shrinkage.

In this technic: 1. No expansion of the wax pattern is obtained. 2. Little thermal expansion of the investment is obtained (0.09 per cent if the casting is made at 160 C. [320 F.], and an actual shrinkage of 0.11 per cent is encountered if the casting is made in a mold at room temperature). 3. Castings are from 1.15 to 1.35 per cent undersize. Castings made by this method have smooth fine surfaces. The best casting of a series is here shown on a die in Figure 8. It demonstrates the inaccuracy resulting when the variables in the casting process are not rationally considered in a technic.

### 2. HIGH HEAT METHOD OF ELIMINATION

The technic employing a high heat method of elimination has the following essential steps:

1. The wax pattern is chilled with room temperature water; washed with 50 per cent alcohol and water, and dried and dusted with powdered graphite, the excess being brushed off. 2. A fine inner investment is mixed with distilled water at room temperature. Small wax rings are used to confine the inner investment around the pattern. 3. A coarse outer investment is mixed in the proportion of 4½ parts of investment to 1 part of water. 4. The inlay is cast when the center of the mold is a cherry red.

The author[4] says that he chills the pattern with room temperature water and, on the same page of his paper, says, "You have noticed that the wax pattern has

been kept at room temperature throughout the different steps, to avoid warpage." It has been pointed out in the discussion of the low heat method that one cannot chill the wax pattern with room temperature water and have the pattern assume that temperature. Ice water must be used, and then before the pattern could be carved or burnished, it would be at mouth temperature.

high for any dental investment. A combination of 10 per cent plaster of Paris and 90 per cent silica will expand to slightly over 1 per cent under these conditions, but this investment is too weak for practical use. The thermal expansion curve of such an investment is given in Figure 9.

The inlays cast by this method would by calculation be from 0.3 to 0.5 per

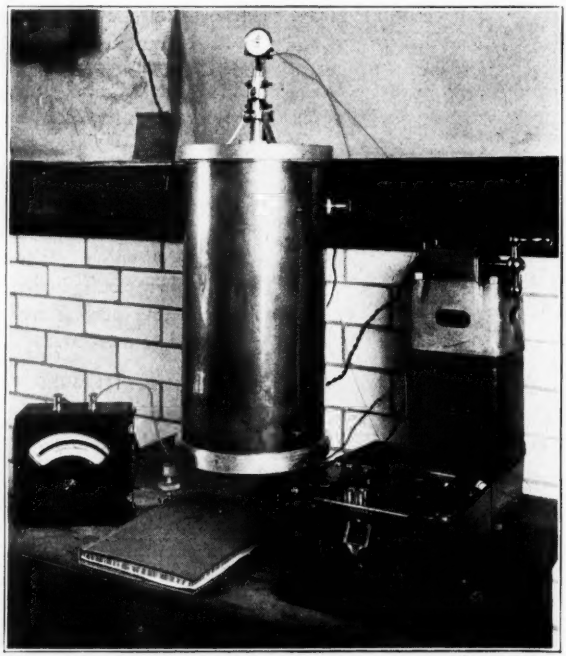

Fig. 7.—Fused quartz tube expansion apparatus and auxiliary equipment.

This technic depends entirely on the expansion of the investment to compensate for the gold shrinkage. We have found no investment on the market that has sufficient thermal expansion for complete compensation of the 1.25 per cent shrinkage of the gold; 0.9 per cent expansion at a bright red heat is extremely

cent too small but will be much closer to fitting than those cast by the low heat method and much more accurate than the cold mold casting.

Figure 10, left, shows the fit of typical inlays cast by this technic.

The author of this technic uses a double investment, and prefers to paint the wax

pattern with graphite and to confine the inner investment with a small wax ring; but these steps are not necessary: equally good results can be obtained without them. The addition of a controlled wax expansion step to this technic is necessary to compensate completely for shrinkage of the gold.

The Ransom and Randolph Co. outline a similar technic for use with their green investment. In this case, a heavy mixture of the investment is placed over the pattern, forming a layer about one-eighth inch thick. After setting, the coated pattern is moistened and the ring filled with a thinner mix of the same investment. When the setting is complete, the mold is heated slowly for fifteen minutes and then at an increased rate to a bright red heat and cast.

The process depends on the complete utilization of the setting and the thermal expansion of the investment, a difficult feat with the ordinary types of laboratory equipment. Castings made by this method were found to fit the steel die fairly well, with indications of slight shrinkages along the margins. (Fig. 10, right.)

### 3. Rapid Elimination of Wax Pattern (Knapp Technic)

Rapid elimination of the wax pattern (Knapp technic) is a popular one, introduced by K. W. Knapp, which has for its chief advertised merits that it is quick, accurate, and standardized. It is quick—quicker than most other technics. As for its accuracy and standardization, we will let the profession decide.

The technic as outlined is as follows: 1. The sprue is placed near the center of the pattern. 2. The pattern is cleaned with Ivory soap and water. 3. Knapp investment No. 1 is painted on the pattern in a layer about one-eighth inch thick. 4. Knapp investment No. 2 is poured around the cherry, which has been allowed to set. 5. The ring is placed in a Knapp eliminator as soon as the investment is completely set and heated from six to eight minutes. 6. The inlay is cast on a Perfection casting machine.

The thermal expansions of the two most recent Knapp investments were determined. (Fig. 11).[11] The graphs show that the maximum expansion of No. 1 investment is 0.12 per cent at 130

Fig. 8.—Casting made by low heat elimination method.

11. The data presented in this paper on these two investments as well as those on other proprietary products were obtained by testing samples purchased in the open market between June 15 and Oct. 1, 1929. They represent the properties of these individual samples, and while they are correct for the package tested, they must not be regarded as the uniform values which the particular manufacturer is striving to produce. Data from the manufacturer's research laboratory covering all packages as marketed will be more appropriate for use by the dentist or dental school. For this reason, the data and curves given in this and other reports should not be used for advertising purposes. The manufacturer without proper equipment to make necessary tests cannot be sure that his factory control is adequate to guarantee compliance with rigid standards.

C. (266 F.), and, at any higher temperature, the material will shrink. At 375 C. (707 F.), it is back to its original dimensions, and at any temperature higher than 375 C. (707 F.), it is smaller than its original dimensions. At 600 C. (1112 F.), this amounts to 0.4 per cent.

The graph of No. 2 investment shows a maximum expansion of 0.11 per cent at 125 C. (257 F.). At 750 C. (1382 F.), it is again back to its original size. At 600 C. (1112 F.), it is 0.05 per cent longer than originally.

TABLE 1.—KNAPP RAPID ELIMINATION TECHNIC (LOW FLAME)

| Time Minutes | Temperature of Mold Degrees C. | F. |
|---|---|---|
| Start | 25 | 77.0 |
| 1 | 75 | 167.0 |
| 2 | 106 | 222.8 |
| 3 | 92 | 197.6 |
| 4 | 92 | 197.6 |
| 5 | 92 | 197.6 |
| 6 | 92 | 197.6 |
| 7 | 92 | 197.6 |
| 8 | 92 | 197.6 |
| 9 | 104 | 219.2 |
| 10 | 136 | 276.8 |
| 11 (Flame out*) | 259 | 498.2 |
| 12 | 366 | 690.8 |
| 13 | 378 | 712.4 |
| 14 | 352 | 665.6 |
| 15 | 314 | 597.2 |

*The continued rise in temperature after the flame was shut off is probably due to the residual heat in the eliminator. Another mix was then made using a higher flame, both eliminations being continued for twelve minutes.

By a comparison of these graphs, we can easily see that No. 1, or inner investment, shrinks more and faster than No. 2, or outer investment. This is probably due to No. 1 investment's high plaster content, as the author[5] states, "The first investment (No. 1) should have a sufficiently high plaster content to withstand the pressure exerted by the water boiling through it in eliminating the wax. The outer or second investment should be composed of a material having high heat resistance."

After the thermal expansion curves of both investments were obtained, it was necessary to determine the temperature (especially the minimum temperature) which the core of the mold attained in eliminating the wax as directed. A thermocouple was embedded in the inner investment and surrounded with the outer investment in a very thin ring. (Fig. 12.)

TABLE 2.—KNAPP RAPID ELIMINATION TECHNIC (HIGH FLAME)

| Time Minutes | Temperature of Mold Degrees C. | F. |
|---|---|---|
| Start | 25 | 77.0 |
| 1 | 50 | 122.0 |
| 2 | 75 | 167.0 |
| 3 | 92 | 197.6 |
| 4 | 92 | 197.6 |
| 5 | 87 | 188.6 |
| 7 | 75 | 167.0 |
| 8 | 123 | 253.4 |
| 8½ | 173 | 343.4 |
| 9 | 247 | 476.6 |
| 10 | 366 | 690.8 |
| 11 | 483 | 901.4 |
| 12 | 577 | 1070.6 |

The ring was heated at the slowest rate at which the wax could be eliminated. Table 1 shows the time and temperature relations which existed under these conditions.

On the thermal expansion curve for investment No. 1, we find the maximum expansion occurred at 130 C. (266 F.). From the low flame temperature table, we can tell how to eliminate accurately by this technic and to obtain the maximum expansion.

A casting was made using a low flame on the eliminator, adjusted so that the temperature would not go over 200 C. (392 F.) in twelve minutes. By this procedure, we can get 0.12 per cent expan-

sion, which is all that is possible with this investment. This is far removed from approximately 1.25 per cent required to compensate for the shrinkage of gold, but how many dentists obtain this optimum condition by either accident or design?

Another inlay was made, the temperature in the mold being allowed to reach 600 C. (1112 F.) in twelve minutes. At this temperature, the investment shrinks approximately 0.4 per cent. Pictures of the two inlays are submitted. (Fig. 13.)

A practical demonstration of the shrinkage of Knapp No. 1 investment is submitted. A model of No. 1 investment was made by the indirect method. A wax pattern was made on it, and the whole invested in "pyramold" No. 1, an investment known to have a rather smooth thermal expansion up to 850 C. (Fig. 14.) The casting was made at 700 C. (1292 F.).

The model should shrink —0.4 per cent. The surrounding investment should

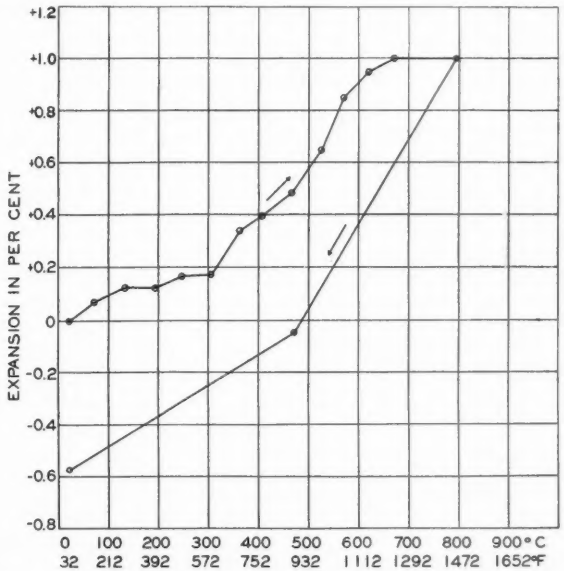


Fig. 9.—Thermal expansion of investment containing 10 per cent plaster of Paris and 90 per cent silica. Rate of heating: From 25 to 300 C., two hours; from 300 to 800 C., two hours. Powder, 100 gm.; water, 42 c.c.

The surfaces are smooth but the dimensions are too small. It is very easy to distinguish the one which had the most shrinkage, i.e., the one cast at 600 C. (1112 F.).

We do not believe it possible to cast an inlay accurately using the Knapp technic and materials. In our hands, the results have been uniformly poor.

expand +0.85 per cent, this leaving a space between the model and surrounding investment. The castings made demonstrate the truth of the statement. (Figs. 15 and 16.)

This casting method has been spoken of as being accurate and standardized. Can a method be considered as such when the temperature of the mold is unknown

at all times; the physical properties of the investments are unknown and in this case happen to shrink, and no effort is made to compensate (by expansion of the wax and investment) for the shrinkage of the gold in casting? Perhaps we should add that one is asked to pay for the special appliances used and for a personal course of instruction in its use.

### 4. THE WATER BATH TECHNIC

The use of a water bath to expand wax patterns for inlays was first suggested by Van Horn.[12] A picture of his equipment is shown in Figure 17. The method has recently been slightly altered (the ring has been completely submerged; Van Horn may not have had it entirely under the water) and incorporated into other technics which have been widely advertised and heralded as new and profound discoveries. The principles are identical and the practical applications are altered in no essential detail.

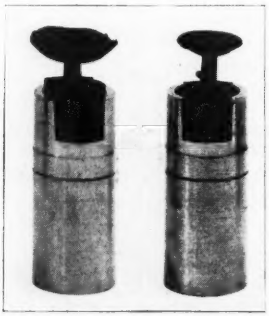

**Fig. 10.—Left: Casting made by high heat elimination method without wax expansion. Right: Casting made by investment expansion only.**

This technic can be accurately controlled and consistent results can be obtained, when it is carefully standardized to suit the laboratory conditions of the individual practitioner.

The essential features are: 1. The pattern is washed with soap and water. 2. Definite proportions of water and investment are used. 3. The investment must be slow setting (from twenty to thirty minutes) to allow the wax sufficient time to expand. A commercial retarder must be added to most proprietary investments. 4. The investment used on the cavosurface is mixed to a putty-like consistency and vibrated into place on a mechanical vibrator or lathe. The other surfaces of the pattern are painted. 5. Both ends of the ring are sealed and it is placed in the water bath at 43 C. (109.4 F.) for inlays and at from 44 to 45 C. (from 111.2 to 113 F.) for a full cast crown until the investment has completely set. (In actual practice, these temperatures are governed by the prevailing conditions, and the exact temperatures used must be worked out by the practitioner.) 6. The wax is burned out by carbonization up to 350 C. 7. The casting is made immediately. 8. In the water bath method as outlined by Furrow,[13] no mention is made as to what temperature the wax is when it is in adaptation to the die or cavity. Obviously, we cannot start to expand from any one of many temperatures and expect to get the same results.

We have pointed out that if the pattern is made by the direct method, its adaptation temperature will be about 36 C. (96.8 F.). If made by the indirect method, it will be at room temperature, which may be from 20 C. (68 F.) to 40 C. (104 F.), according to the climate and season. If the pattern fits at 20 C. (68 F.) and is placed on the water bath at 43 C. (109.4 F.), there would be a

12. Footnote 6, first reference.

13. Footnote 6, third reference.

range of 13 degrees C. (23.4 degrees F.), which would expand the wax about 0.91 per cent. If the pattern was fitted at 40 C. (104 F.), there would be an expansion range of only 3 C. (5.4 F.). In this case, the wax would be expanded only 0.21 per cent (0.07 per cent being the increment in length of the wax for each degree centigrade). If castings are made from the two patterns at the same temperature, the end products would vary in dimensions by about 0.7 per cent.

While it is convenient to own an exorbitantly priced water bath, it is not necessary, and equally good results can be obtained with a thermometer and an enamelware pan.

Again, as good results can be obtained by painting on the investment as by vibrating on a heavy mix, and it is less complicated.

A picture of two inlays cast by this method using proper temperatures for wax and investment expansions are shown in Figure 18. They are very smooth and have not been pickled or burnished. They will require little finishing or burnishing.

Care must be taken not to vibrate the case before the plaster is completely set, as the jarring brings the water to the surface of the wax pattern, causing surface roughness.

Figure 19 shows one extremely rough casting caused by running a mechanical mixer on the same bench with the water bath, and a control which was treated at the same time in a separate bath which was not subject to vibration.

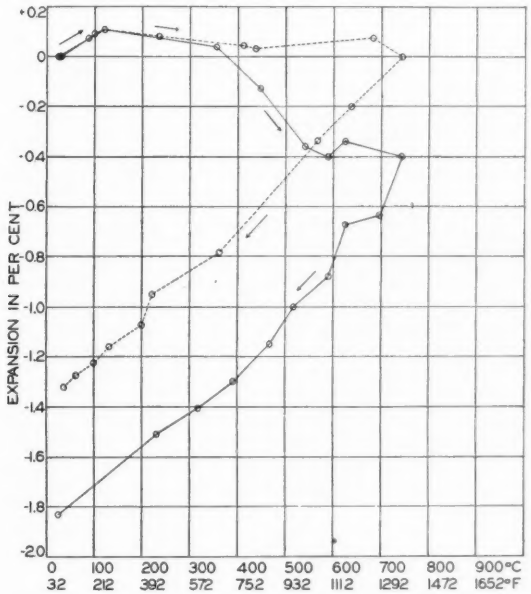


Fig. 11.—Thermal expansion of Knapp investments, outer and inner, 1 and 2. Rate of heating: From 25 to 750 C., thirty-eight minutes. Outer, No. 2, broken line; inner, No. 1, solid line.

The Ransom and Randolph Co. have a modified water bath technic in which the painted pattern is immersed in the bath. This variation was tried and found to be satisfactory when wax expansion and investment expansions are correctly arranged.

No mention is made as to what temperature the wax must be when fitted to the cavity. It may be pointed out again that one cannot expand wax from any temperature up to from 35.56 to 36.67 C. (96 to 98 F.) and get the same results. The picture shows an inlay made by this technic in place on a die. (Fig. 20.) The wax was made to fit at 25 C. (77 F.), immersed in the water bath at 35 C. (95 F.) and cast at about 700 C. (1290 F.). The thermal expansion curves for R. & R. green investment with varying water to plaster ratios are shown in Figure 21.

5. MEYER-MAVES CASTING TECHNIC

The Meyer-Maves casting technic was originally presented in the August, 1928, issue of THE JOURNAL by H. A. Maves, who acknowledges that he was presenting the deductions and technic of F. S. Meyer. An almost identical technic was presented by F. S. Meyer nine months later in the May, 1929, issue of THE JOURNAL.

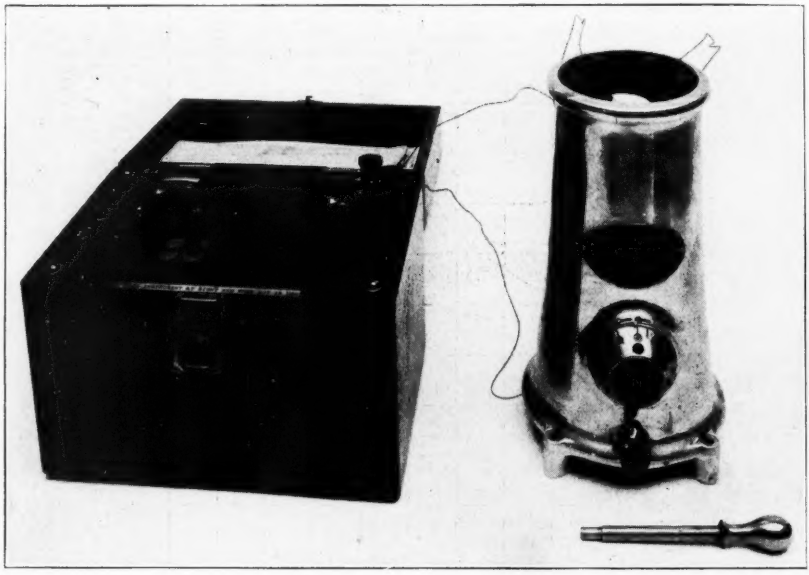

**Fig. 12.—Equipment used in determining burnout temperatures in Knapp rapid elimination technic.**

The technic as outlined by Dr. Maves was essentially as follows: 1. A slow setting outer investment of lower plaster content (Taggart's) is mixed with water at 54 C. (130 F.), and the bowl placed in an oven at 54 C. (130 F.) until needed. 2. A slow setting high plaster

inner investment (Meyer and Maves investment) is used to paint the wax pattern, all materials being at room temperature. 3. The outer investment is poured around the painted pattern, and placed in the wax expander for forty minutes. 4. The wax is boiled and steamed out in a special boiler after the setting is complete. 5. The mold is placed in an electric eliminator until all moisture (as evidenced by vapor on a cold mirror) is removed and the casting then made. The process as outlined is complicated and the details given too vague to be followed easily.

Fig. 13.—Castings made by the Knapp rapid elimination technic. Left: Cast at 1112 F. Right: Cast at 392 F.

Some improvement in detail is shown in the material presented by Dr. Meyer. The variations in this second presentation are: 1. The outer investment (in this case Dr. Meyer's outer investment) is mixed with water at 76 C. (170 F.). The temperature of the resulting mix is about 46 C. (115 F.). 2. The inner investment used is Dr. Meyer's inner investment. 3. The mold, after the outer investment is poured, is placed on top of the wax eliminator, where the temperature should reach 48 C. (120 F.) before setting. 4. The casting is made after the mold has been placed in an oven at 425 C. (800 F.) for sixteen minutes.

From the outline (if we consider the latest presentation), it is evident that the technic is a combination of (1) wax expanion by the hot investment method; (2) quick elimination of wax, and (3) moisture elimination at a comparatively low, but indeterminate, temperature.

Dr. Meyer says, "If the investment is mixed at a temperature 50 degrees above room temperature and maintained at that temperature until it sets, the wax model will be expanded 1 per cent." If he really did this in his technic, his direct wax model might be expanded from 0.56 per cent if the room temperature is 65 F. (18 C.) to over 1.5 per cent if the thermometer hovers around 90 F. (32 C.) as it may during the summer months. Room temperature near 77 F. (25 C.) would give the 1 per cent expansion indicated. As has been previously mentioned in the discussion of other technics, it is not practical to adapt wax to a cavity in the mouth at other than mouth temperature. In this case, more uniform results would be obtained if the wax were expanded by heating at a fixed temperature rather than to a temperature fluctuating with the room conditions.

The wax is eliminated by placing the ring in a special boiler which has a water compartment and a sealed off space for the ring so that steam and water can boil through the investment, carrying the wax out, but no better results are obtained than by the carbonization method. It is a superfluous step—entirely unnecessary—but appears to be harmless.

Thermal expansion curves of the four investments used are shown in Figures 22 and 23.

Maves used S. S. White investment Compound No. 9 (especially compounded for this technic) as his inner investment. Taggart's material was used for the outer investment. They show practically the same expansion curves, i.e., an increase in length followed by a shrinkage and that by a second expansion. Obviously, one must be careful not to cast at a temperature at which the investment shrinks. As nearly as his paper can be interpreted, castings were made at about 150 C. (302 F.). At this temperature, White's No. 9 investment shows an expansion of 0.15 per cent. This, plus the variable wax expansion obtained, might, under optimum conditions, give a perfect-fitting inlay. The curve for this investment shows that accidental overheating of the mold would be troublesome owing to the excessive shrinkage of the material at temperatures between 350 C. (662 F.) and 600 C. (1112 F.).

The expansion curves for Dr. Meyer's investments are similar to the others and indicate that similar results may be expected with them. If a mold were through accident allowed to reach the temperature of the eliminator, 426 C. (800 F.), and the casting made at that temperature, the conditions would be the worst which could be obtained with these materials, as that temperature corresponds closely with the maximum shrinkage observed. This could occur within a very few minutes.

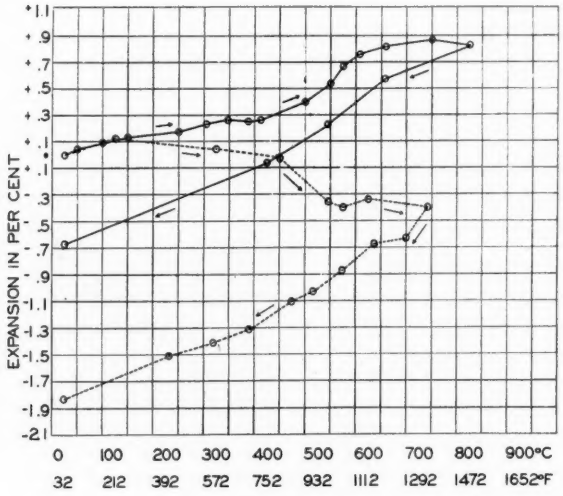


Fig. 14.—Comparative thermal expansion curves of Knapp investment No. 1 and "pyromold" investment No. 1. Knapp No. 1, broken line; "pyromold," No. 1, solid line.

Dr. Meyers also says, "There are several reasons for using a double investment: to get the heaviest possible high plaster content mixture, which cannot be poured, next to the wax pattern; to get a more porous outer investment, which will facilitate the escape of air in casting, and to get a lower plaster content outer investment which will withstand the high heat of elimination without disintegra-

tion." At another point in his paper, he explains his choice of an inner investment by saying, "In the quick elimination method, a relatively large percentage of plaster is used, producing a much harder mold which offers a great deal of resistance to the shrinkage of the gold. This also expands more on heating because of its higher plaster content. When the high plaster content investment is used, the wax must be eliminated before the moisture because the mold will not stand the high heat necessary to carbonize the wax."

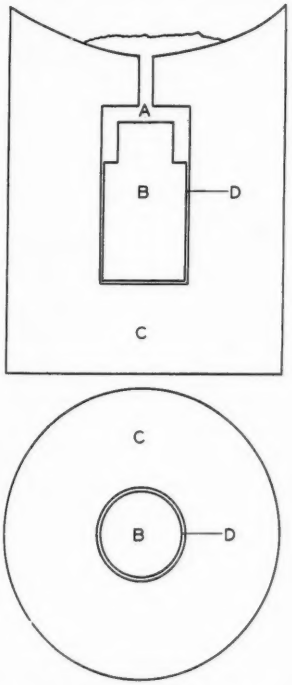


Fig. 15.—Effect of casting on a model of Knapp No. 1 investment surrounded by "pyromold" No. 1 investment. *A*, casting. *B*, Knapp No. 1 model. *C*, "pyromold" No. 1. *D*, large fins on casting due to shrinkage of the model and expansion of the outer investment.

It may be pointed out in considering these statements that: 1. If the high plaster content inner investment will not withstand high temperatures, there is little necessity of having an outer investment which will. 2. The available data on investments indicate that high plaster investments do not expand more than high silica types, but, on the contrary, show greater tendencies to shrink. (See previous reference to the work of R. L. Coleman.) 3. No data are offered in support of the statement that a high plaster investment was used on account of its resistance to the shrinkage of the gold. In view of the tendencies this type of investment has to shrinkage and its low resistance to heat, some data should be presented in support of a statement of this kind.

Fig. 16.—Castings to illustrate differential dimensional changes in Knapp No. 1 and "pyromold" No. 1 investments. Left: Casting, showing heavy fins produced. Center: Casting still in place on model. Right: Typical model before investing.

The process as described is unnecessarily complicated and requires too much attention to detail at frequent intervals to be very practical for the average practitioner. The use of a single heat-resisting investment which has a continual expansion rather than the two investments described would make the process more adaptable for general use. One of

the outstanding features of the paper was the very apparent effort put forth to convert the profession to the use of the special equipment described.

The process will, if properly handled and standardized (the technics given are, as shown above, far from a standardized condition), give satisfactory results, but considerable time and effort are needed to translate the method as given into a workable laboratory scheme. Equal or better results can be produced by much simpler methods. Castings made by the process on a day when room temperature was 28 C. (82.4 F.) are shown in Figure 24.

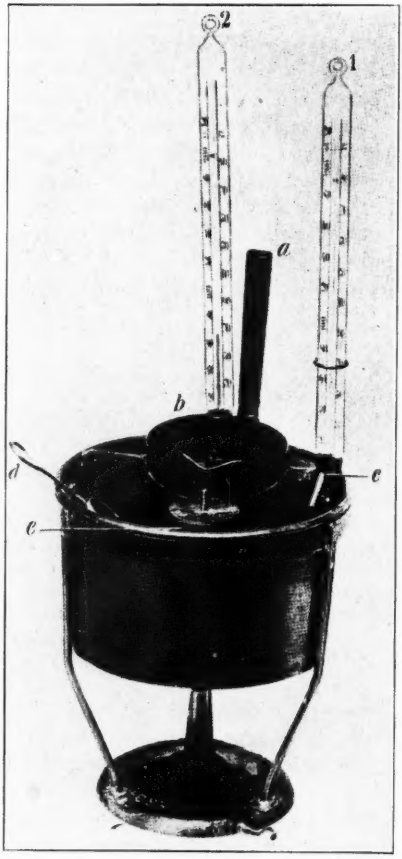


Fig. 17.—Parts assembled, with various articles in position in the racks. *a*, mixing spatula. *b*, mixing cup with cover in position. *c*, sprue wire. *d*, water measure. *e*, flask with cover in position. (Van Horn's original wax expansion equipment [from Dental Cosmos, 1911]).

### 6. THE SEMIDIRECT METHOD

The semidirect method consists of (1) taking an impression of the tooth or die, using a band and modeling compound;

Fig. 18.—Inlays cast by technic involving wax expansion in a water bath.

(2) filling this impression with a very heavy mix of plaster of Paris, and (3) taking another indirect impression of the plaster model, and pouring that up in plaster of Paris. This process is undoubtedly repeated the number of times necessary to compensate for the gold shrinkage, the plaster of Paris expanding on setting and each resulting model being slightly larger than its predecessor. The number of steps will depend on the technic used, i.e., the consistency of the plaster of Paris, or the investment used. It

is possible to compensate fully for the gold shrinkage by this method, but there is a slight loss of detail due to the repeated reinvestment.

An inlay made by this method is shown seated on a steel die (Fig. 25). In this particular case, the plaster model on which the wax pattern was made was stepped up twice, giving an expansion of about 0.45 per cent. A high expanding high heat investment was used; otherwise, more plaster models would have been required. This inlay would not go to place on the plaster model from which its pattern was taken.

This method is hardly practical when full compensation is desired. It is too intricate, and several more desirable methods have been developed.

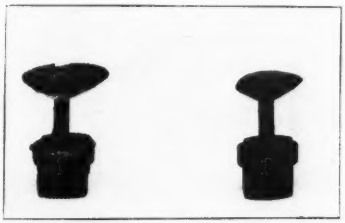

Fig. 19.—Effects of vibration on the surface of castings produced by the water bath wax expansion technic. Left: Casting produced after bath had been subjected to considerable vibration during the setting period of the investment. Right: Control sample made without vibration.

### 7. THE WHIP-MIX TECHNIC

The essential features of the Whip-mix technic are as follows: 1. Definite proportions of water and investment are weighed in a special balance accompanying the investment. 2. The investment is mixed in a special mechanical mixer for at least thirty seconds, making seventy-five or more revolutions of the handle. 3. The pattern is heavily painted and the remainder of the mix poured around it. 4. It is allowed to set for thirty minutes. 5. The wax and moisture are eliminated in a special furnace, the low heat being used for ten minutes, then full heat. 6. The case is cast when the periphery of investment is light, the center being dark.

A thermal expansion curve of this investment is shown in Figure 26. Notice that the investment has a very small thermal expansion up to 500 C. (932 F.). According to the directions, a mold is approximately 450 C. (842 F.) when cast. At this temperature, the mold would be expanded 0.1 per cent. Since no wax expansion is provided, the castings should be approximately 1.1 per cent too small. The investment evidently contains graphite. This, with the weighing device, strongly suggests an imitation of certain parts of Taggart's materials.

Fig. 20.—Casting made by the R. & R. technic, combining wax and investment expansion.

An inlay cast according to these directions will not be accurate. Figure 27 shows the accuracy obtained.

The latest technic advocated for the Whip-mix technic is one for mesioclusodistal inlays: "The 'MOD' inlay has always been a thorn in the side of the casting technician. The following technic is given mainly to overcome the shrinkage at the gingiva."

This technic differs from the regular one in that: 1. Water at from 140 to 160 F. (60 to 71 C.) is used in mixing the investment. 2. A heavy inner warm mix is painted on the pattern. 3. An outer warm mix is poured around the painted pattern when the gloss on it has disappeared. 4. The procedure as outlined in the original technic is followed in the burnout and casting.

Two special mechanical mixers are recommended, together with a special inlay furnace, casting machine and other special accessories.

The directions for the wax expansion are indefinite. A 20-degree F. range in temperature for the water is indicated. This might easily result in variations in the dimensions of different castings as great as 0.35 per cent. The directions state that the water temperature can "conveniently be ascertained by placing the hand in pan of water." This procedure could not be considered standardized.

When room temperature is 22 C. (71 F.) and the mixing water at 71 C. (160 F.), the resulting mix will be 42 C. (107.6 F.). If the inlay patterns are in adaptation at room temperature, the resulting inlay will fit, but if they fit at mouth temperature, they will be

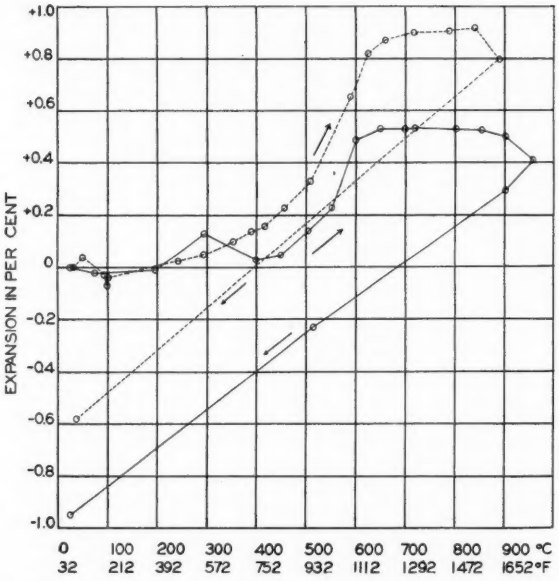


Fig. 21.—Thermal expansion of Ransom & Randolph green investment. Rate of heating: from —30 to 300 C., two hours; from 300 to 600 C., one hour; from 600 to 900 C., one hour. Powder, 100 gm.; water, 32 c.c., broken line; powder 100 gm.; water 50 c.c., solid line.

0.8 per cent too small. The necessity of operating in rooms at temperatures other than 71 F., the indefiniteness of the water and investment mix and the meager expansion of the investment are factors in rendering this technic subject to definite criticism. Figure 28 shows an inlay cast by this technic.

The comparison of these technics has shown that no process can consistently give satisfactory cast inlays unless it takes into consideration the variables entering into the casting process and counterbalances the shrinkage of the gold by careful combination of the factors producing an enlarged mold. Almost any technic, no matter how inadequate or unbalanced, may at some time or another, through a lucky and accidental combination of favorable conditions, produce an inlay which will be satisfactory. Neglect of the fundamental factors involved must result in an exceedingly large proportion of failures.

It is unfortunate that so many technics have been presented to the dental profession which have failed to give careful consideration to the scientific basis for the casting process. These are almost universally based on the accidental production of a fairly good inlay. They give no definite standards or values for the variables entering the process and must of necessity be regarded as attempts at imitations of another's luck rather than scientific attainments. A careful review of the various casting processes would indicate that, if reasonable attention is given the variables in the casting process, satisfactory castings can be the rule instead of the exception. Any process which embodies the following general

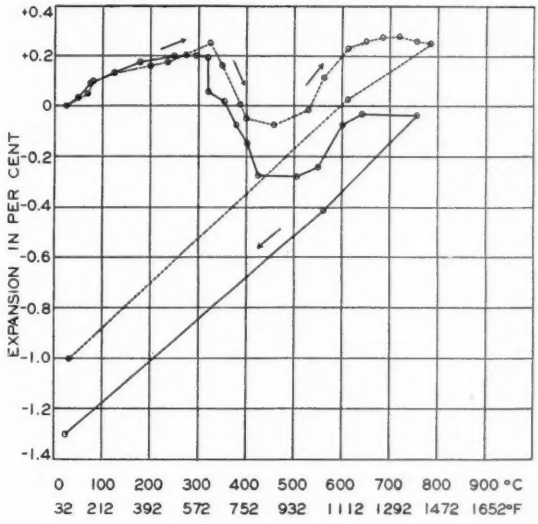


**Fig. 22.—Thermal expansion of S. S. White investment compound No. 9 and Taggart fluid inlay investment. Rate of heating: From —25 to 325 C., one hour; from 325 to 500 C., one hour; from 500 to 750 C., one hour. S. S. White No. 9, solid line; Taggart investment, broken line.**

principles is capable of adaptation to any ordinary laboratory conditions:

1. An investment should be chosen which has a smooth thermal expansion curve showing a continued increase in length from the temperature at which the moisture is eliminated up to a bright red heat. The temperature expansion relations must be known. Manufacturers should supply such curves for their various investments together with data on

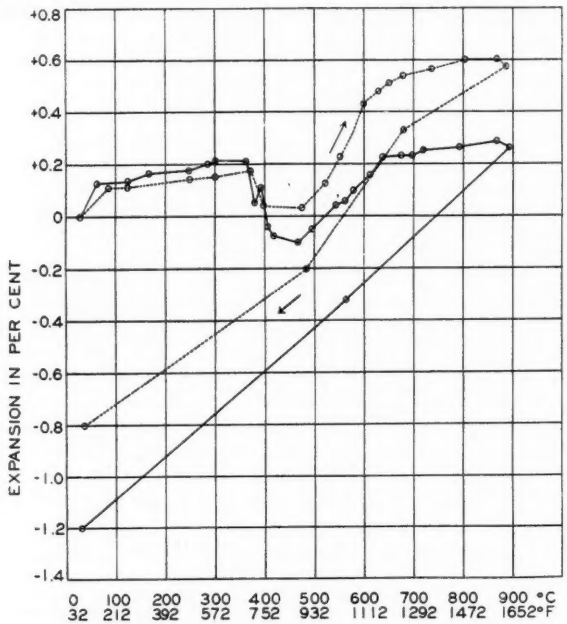


Fig. 23.—Thermal expansion of Dr. Meyer's investments. Rate of heating: From 30 to 350 C., one and one-half hours; from 350 to 650 C., two hours; from 650 to 900, one hour. Inner investment, solid line; outer investment, broken line.

other physical properties as proof of their usefulness rather than using extravagant claims, unsupported by scientific tests. Certain groups seem to have fallen into this type of propaganda, and whether intentional or not, the exploitation of the profession is very evident. Dentists must cease to be victims of sales propaganda if they are to be true to their profession. We are sure you will excuse this harsh statement, but to us, now familiar with the properties of materials and methods of advertising, and as representatives of your profession instructed to bring to you facts without fear or favor, there appears to be no other choice for us in this presentation of the situation. Truth in advertising must be insisted on regardless of whether the "ad" is a "paid for ad" or an "ethical endorsement" in the reading pages disguised as a scientific paper.

2. A wax should be selected whose thermal expansion characteristics are known and whose other physical charac-

teristics are such as to permit the making of satisfactory patterns. In the event no expansion data are available, it is fairly safe to assume a value for the thermal expansion of any inlay waxes since their expansions are rather similar.

The value previously mentioned of 0.07 per cent a degree centigrade or 0.04 per cent a degree Fahrenheit will serve for preliminary calculations.

3. The casting shrinkage of the golds being approximately 1.25 per cent, the three factors which may be made to balance this shrinkage are to be so controlled that their total value will equal 1.25 per cent. The exact way in which the factors are to be combined is largely a matter of individual choice.

At the present time, the actual extent to which the setting expansions of investment may be used to compensate for casting shrinkage is unknown. These setting changes enter into the process to some extent, but from the available data, not completely. Fortunately, the whole possible change is small and can be counterbalanced in the final practical balancing of the other two factors.

Neither wax expansion nor investment expansion alone is sufficient for the complete balancing of gold shrinkage.[14] It is more reasonable to provide most of the compensation by investment expansion since wax is liable to distortion at high temperatures. The combination of the two factors may be easily calculated. A wax pattern, adapted to the cavity at mouth temperature, is expanded to some definite temperature, not above the softening point of the wax, by any one of several methods:

1. The pattern may be placed in an oven at the desired temperature, and, when it has had time to come to the oven temperature, can be coated with investment at that temperature and allowed to stand until the setting is complete.

2. It may be coated with a slow-setting investment at room temperature, and the ring filled and placed in a water bath at a fixed, known temperature.

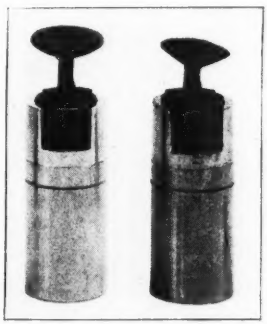

Fig. 24.—Castings made by the Meyer-Maves technic.

Fig. 25.—Casting made by the semi-indirect method (Solbrig).

14. Since this paper was first presented, investment materials have been developed which have thermal expansions greater than 1.25 per cent. Details will be given at a later date.

3. The coated pattern may have a mixture of investment and hot water at a known temperature poured around it to produce the expansion. Of the three methods, this requires the greatest care in manipulation and is the most affected by variations in room temperature.

The maximum temperature reached by the wax prior to the setting of the investment being known, the amount of compensation obtained can be calculated approximately. If the temperature is 45 from the thermal expansion curve of the investment.

The trial casting temperature and temperature for wax expansion having been determined, preliminary casts are made under carefully controlled and recorded temperature conditions. Underexpansion or overexpansion is eliminated in the product by varying either the temperature of the wax expansion or the casting temperature. The exact temperatures which give satisfactory results in one

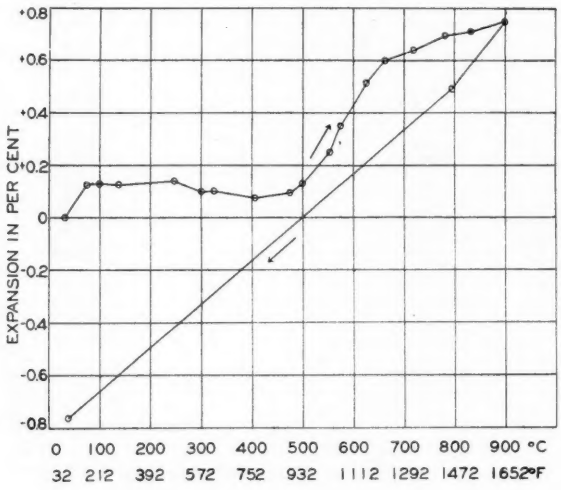


Fig. 26.—Thermal expansion of Whip-mix inlay investment. Rate of heating: From —30 to 500 C., two hours; from 500 to 900, two hours. Powder, 100 gm.; water, 52 c.c.

C., the method would be as follows: 45 to 37 (body temperature) = 8 degrees; 8 × 0.07 per cent (expansion for each degree) = 0.56 per cent wax expansion.

The wax expansion is subtracted from 1.25 per cent, leaving, in this illustration, 0.69 per cent expansion to be obtained in the investment by heating. The trial casting temperature may then be read

laboratory will seldom do so in another, and the balancing operation described is necessary in adapting any process for the use of an individual practitioner.

## CONCLUSIONS

1. The thermal expansion characteristics of the investments used are necessary factors in the success of any inlay casting process and should be available to the operator.

2. Few of the casting investments now available are of good quality. The majority of the current investments tend to shrink through the temperature ranges commonly used as casting temperatures and have insufficient total expansion to be valuable for all-round use.

3. The use of a single investment for painting and to fill the casting ring is desirable. No advantage was shown in any technic studied which would justify using two different investments.

4. Several current technics appear to be advocated primarily as sales promotion schemes to assist in marketing special equipment, too often of doubtful value but always high in price.

5. Satisfactory cast inlays can be consistently made by any process which correctly balances the variables encountered in the casting process.

6. The equipment necessary is dependent on the desires of the individual operator and may range from a combination of an enamel pan, a bunsen burner, a thermometer, a torch and a simple casting machine, to the very elaborate outfits sold in connection with commercialized technics, not one of which has, to our knowledge, been accurately standardized for variations in room temperature, or definitely coordinated with the known physical properties of the various waxes or investments used by the profession. Either type may be made effective if standardized, coordinated and intelligently used.

Fig. 27.—Casting made by Whip-mix inlay technic.

Fig. 28.—Casting made by Whip-mix mesioclusodistal technic.

# "WHY ROOT CANALS SHOULD BE FILLED TO THE DENTINOCEMENTAL JUNCTION"*

By WILLIAM G. SKILLEN, D.D.S., Chicago, Ill.

ALTHOUGH many of the statements contained in Dr. Grove's article, "Why Root Canals Should Be Filled to the Dentinocemental Junction," may be justly criticized on the basis that they lack substantiation, I am particularly interested in the anatomic features on which the technic is supposed to have been based, and will, therefore, limit my discussion to that aspect.

The first thing which I wish to take under consideration is the apical dentinocemental junction. Grove, at first, says that "a definite junction of the dentin and the cementum exists," and immediately continues with the statement: "That there are irregularities in the line of demarcation dividing these two structures is not questioned." Obviously, this is a contradiction, for if irregularities exist, the junction cannot be so "definite" as he leads one to infer from his first statement.

Dr. Grove goes on to say that this definiteness is not questioned by those who have studied sections of root end tissue prepared for this purpose only; but he fails to state who those persons are, and I must acknowledge that I do not know of the existence of any such evidence. I have made sections of teeth expressly for the purpose of studying root canal conditions. As it was deemed imperative that these sections should show the entire root canal, they were cut horizontally and serially. This is the only type of section which will give a view of the entire circumference of the canal from one end of the tooth to the other. Even on the basis of such material, I would not venture to say more than Coolidge[1] already has said, namely, that the apical region of the tooth, and the arrangement of tissues in that region, are most variable. The very complexities of the area make it impossible to tell, except approximately, what arrangement exists. Probably nothing short of many hundreds of reconstructions from such material would enable one to draw any definite conclusions as to the structure of the apical area. This has never been done and it is questionable whether it would be worth the labor it would entail.

Grove further questions the value of sections of material such as Coolidge presented and such as are included here, claiming that they are misleading; that they do not show the complete picture; that they may be of such nature that their details are not recognizable. It is willingly granted that, to a certain extent, he is right. Any section of any material may be misleading to those who

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*A discussion of an article published under this title by Grove, C. J.: J.A.D.A., 17:293 (Feb.) 1930.

1. Coolidge, E. D.: Anatomy of the Root Apex in Relation to Treatment Problems, J.A.D.A., 16:1456 (Aug.) 1929.

are not accustomed to reading them. The interpretation which is placed on them is the important thing from the standpoint of the uninitiated. The details which may be shown are dependent on a number of things and often are lacking. If they are not reasonably clear, such illustrations, of course, should not be used. It is also granted that sections cut parallel with the long axis of the tooth do not tell the whole story. As has been indicated, probably only reconstructions from horizontal, serial sections would do this. The former do illustrate some things. As far as the apical region of the tooth is concerned, they show, at least, that a tremendous variation exists between one tooth and another. Coolidge showed this. If such variation exists between teeth, and if, as Grove himself admits, irregularities occur in the apical arrangement of any one tooth, it is a fair guess that there exists no such general, definite arrangement as Grove would like us to believe, nor one that could be at all counted on even if this were thought necessary.

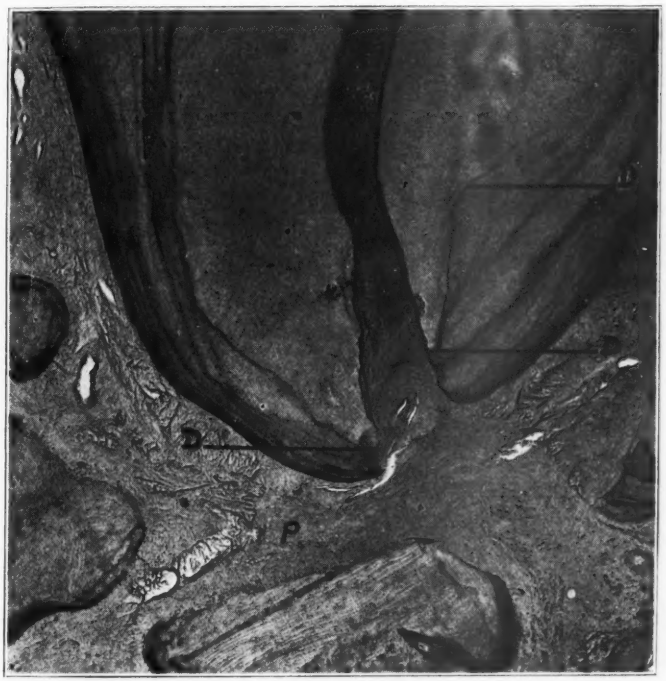


Fig. 1.—*D*, dentinocemental junction. Similarity between the tissue in the canal *C* and the peridental membrane *P* is to be noted.

But attention must be called to the type of evidence which Grove himself offers in support of his theory. This is of exactly the same nature as that which he criticizes—single sections cut parallel

with the long axis of the tooth. Moreover, they apparently represent teeth in which the roots have not been completely formed, not the finished structure which the average dentist is most often called on to treat. The tissues too are torn and distorted, so that their relationships are not clear, and on top of all this, details

**Fig. 2.—*D*, dentinocemental junction. Similarity between the tissue in canal *C* and the peridental membrane *P* is to be noted. (From a 45-year-old individual.)**

of structure are sadly lacking. To offer such material as proof of the general definiteness of a dentinocemental junction is, to say the least, very misleading.

In a previous article,[2] Dr. Grove, referring to this feature, says: "The condition that I refer to is not such as is seen when the cementum has been deposited in the foramen of teeth in middle life or advanced age. This is very irregular and no definite junction can be seen. It will also be observed that a large number of canals extend through this structure into the periapical region, and there is also an intermingling of dentin. One should not confuse these with the apical dentinocemental junction."

Now, as this seems to put a new meaning on the term "dentinocemental junction," it rather behooves Grove to define it. I have as yet been unable to find that he has done so. It must also be asked at what age cementum is deposited in the foramen and multiple canals develop, and if these are characteristic only of middle and old age. As far as I know, no correlation of such structure and age

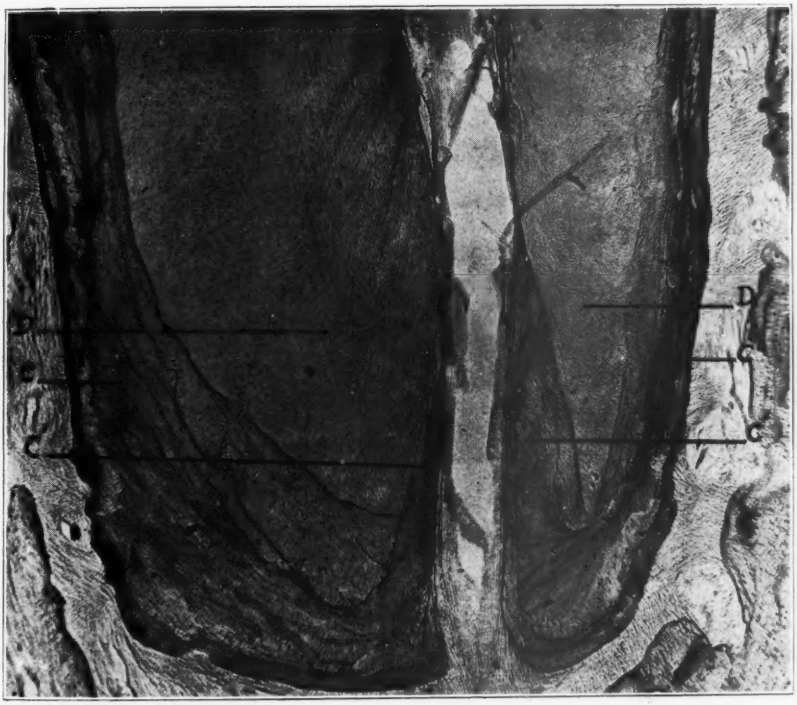


Fig. 3.—*C'*, cementum folding back into canal; *D*, dentin; *C*, cementum. (From a 25-year-old individual.)

2. Grove, C. J.: Method of Pulp Removal to Prevent Periapical Infection, J.A.D.A., 10:93 (Feb.) 1923.

has been made. The work of Hess[3] seems to be the nearest approach to this that there is today, and he shows the presence of multiple canals from a comparatively early age on. But this, after all, is begging the question. Teeth are not treated on an age basis; they must be taken as they come.

is dependent. The next moment, he states that it is only by transformation of the pulp tissue into fibrous tissue that healing occurs.

Obviously, we are again dealing with a contradiction, for if it heals at all, it must possess the essentials for healing. Probably what he means is that it does

Fig. 4.—Slight extension of cementum into canal. *D*, dentinocemental junction.

The second point on which I wish to touch is Dr. Grove's ideas concerning the pulp. He states that the pulp, being an embryonic tissue, lacks certain cell characteristics on which the process of healing not return to its original form. That the character of a tissue may change in the course of healing is not unusual — quite the reverse: such changes occur elsewhere in the body. Nevertheless, this constitutes a healing process and the tis-

3. Hess: Anatomie der Wurzelkanäle.

sue may remain for indefinite periods in this changed condition. Furthermore, there is to be obtained from the literature which are not so different from those which go on in other parts of the body. Orban[4] too has shown that the pulp con-

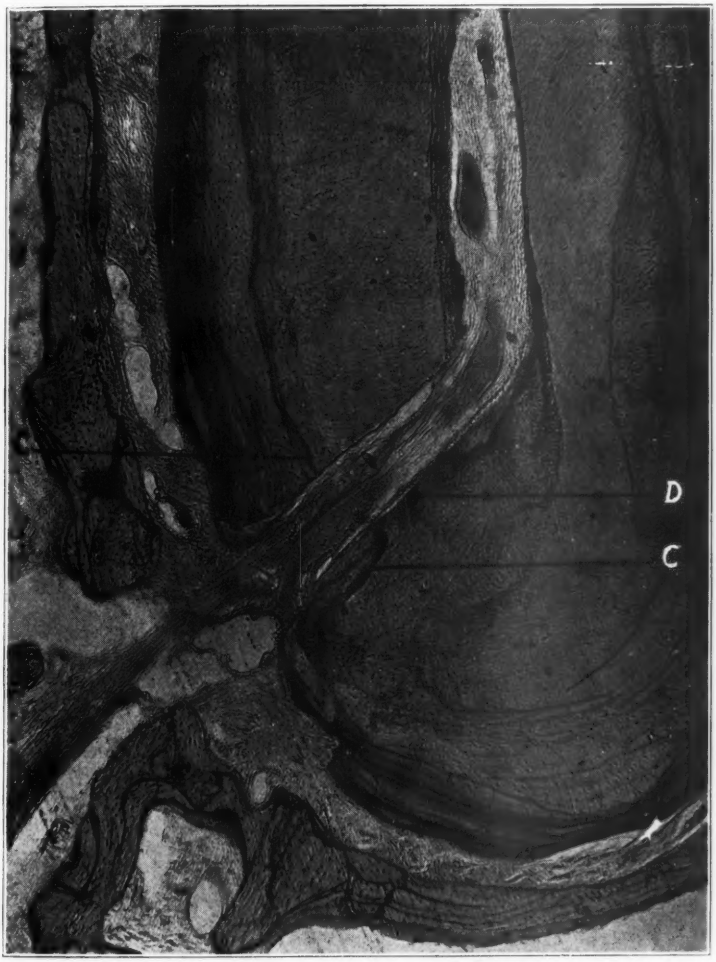


Fig. 5.—Canal opening onto side of root; *D*, dentin; *C*, dentinocemental junction. Similarity between the tissue in the canal and the peridental membrane is to be noted.

some evidence of the favorable reaction, the high degree of resistance of pulp to various forms of irritation, reactions

4. Orban, Balint: Dental Histology and Embryology, Philadelphia: P. Blakiston's Son and Co., Ed. 2, p. 51, 1929.

tains the same specialized defense elements as do other tissues.

But this is neither here nor there. The fact is that, at the present time, no one knows just what lies behind the success or failure of the treated tooth. To say, in either case, that it is due to inherent properties of the pulp or other tissues is simply guessing.

Grove further states that there is a sharp line of demarcation between the pulp and the peridental membrane. As far as I know, this too has never been demonstrated. I have never been able to do more than imagine a difference probably owing to the fact that there are present tissues (dentin and cementum) the formation of which is usually ascribed to pulp and peridental membrane, respectively. Eliminate these structures from view and it frequently becomes impossible to clearly distinguish between pulp and peridental membrane either in the developing or in the adult tooth.

We might lean toward the view that only peridental membrane will form cementum, and pulp form, dentin, if it were not for the fact that frequently other types of tissue besides tubular dentin seem to be the product of the pulp. Numerous instances of the formation of even a cementum-like tissue in relation with what seems to be pulp have been noted in the literature. This may mean, of course, that peridental membrane elements are encroaching on the pulp, but,

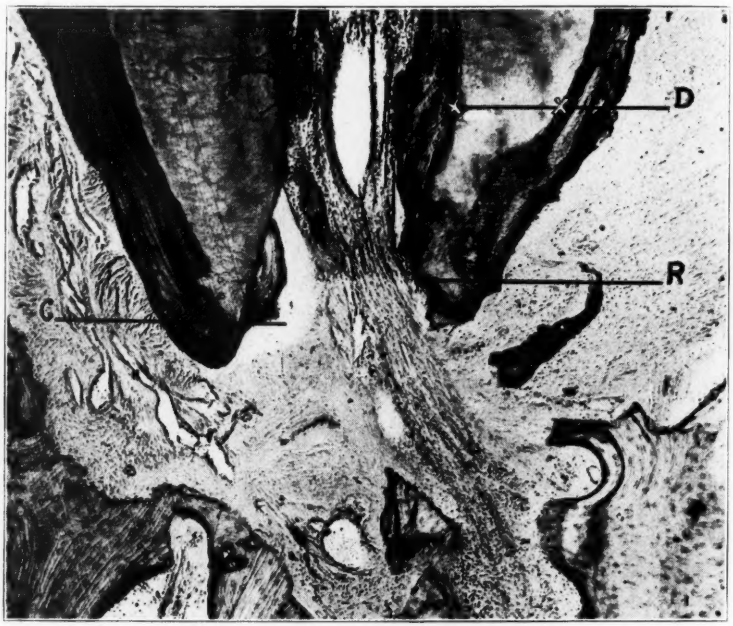


Fig. 6.—*D*, dentinocemental junction; *R*, resorption into dentin; *C*, new formation of cementum on top of old.

at the present time, it is an open question. The fact remains that we cannot say definitely where pulp terminates and peridental membrane begins.

Finally, I wish to say just a word in regard to Dr. Grove's opinion as to the imperviousness of cementum, particularly that in the apical region, and the relation area of the dentinocemental junction, may be permeable (Box, Skillen). This evidence Dr. Grove has apparently overlooked. So long as this stands uncontradicted, it must be given some consideration and the full efficacy of cementum to act as a sealing agent must remain doubtful. Aside from this, there is no

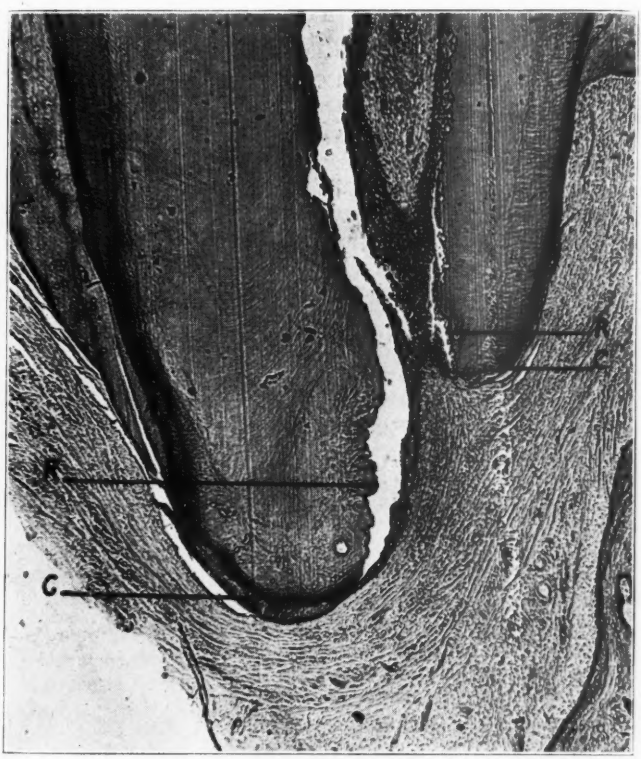


Fig. 7.—Carious root from a 20-year-old individual. *R*, resorption in dentin; *C*, dentinocemental junction.

of infected dentin to failure with respect to the treated tooth.

From the structural and from the experimental standpoint, there is some slight evidence that cementum or, better, the definite proof that previously infected dentin involves the future of the treated tooth. On the other hand, both Kronfeld[5]

5. Kronfeld, Rudolf: Ztschr. f. Stomatol., Vol. 26, 1928.

and Coolidge[6] have shown that dentin possibly in just such a condition may react quite favorably. And although such evidence is very limited at the present time, it also must be given consideration.

In closing, let me call attention to the criticism of this article already offered by Orban,[7] with whose expressed views in general I am in accord, and let me add this statement: All too frequently articles are written in which statements are made which are not substantiated and in which theory becomes fact over night. And nowhere does this appear to be so prevalent as in connection with the root canal problem. As things stand today, there is not one particle of real evidence as to what lies behind success or failure in the case of the treated tooth. Furthermore, there is not one bit of evidence upon which the worth of any one of the numerous technics in use may be satisfactorily evaluated. The thing that we need may lie right in our hands and not be recognized. To seek farther under the circumstances is only uselessly complicating an already complex matter. In the words of Orban, it is time to substitute a few facts for opinions and theories.

Since the preparation of this article, there has appeared in the August (1930) number of THE JOURNAL the reply of Dr. Grove to the criticism of Orban. Attention must again be called to the fact that Grove has again used sections somewhat similar to those which he criticized as being inadequate, and that these, moreover, are considerably lacking in detail and accuracy. For instance, Grove's figures do not show the complete apical foramen. This, in turn, makes it impossible to tell just where the root filling terminated. It may have passed entirely through the foramen. Figures 4 and 5 in his article seem to show that the filling terminates *in cementum, not at the dentinocemental junction,* which is an entirely different matter from the one Grove has been emphasizing. I am quite willing to agree that it may be possible sometimes to fill canals so that the end of the filling may terminate in cementum, but I am not willing to admit that, even in such instances as Grove's Figures 4 and 5 illustrate, the filling necessarily is entirely surrounded by cementum. Neither am I willing to admit that this can be done as a regular thing, nor that, as the evidence now stands, it is at all necessary to do so. There is no proof that "an open foramen can cause infection." Attention is further called to the fact that a much different type of tooth is used in illustrating this article than was used in the previous one. These are adult teeth and not incompletely developed teeth.

6. Coolidge, E. D.: Root Resection as a Cure for Chronic Periapical Infection; Histologic Report of a Case Showing Complete Repair, J.A.D.A., 17:239 (Feb.) 1930.

7. Orban, Balint: "Why Root Canals Should Be Filled to the Dentinocemental Junction," J.A.D.A., 17:1086 (June) 1930.

# A DENTAL EDUCATION AS AN INVESTMENT*

By F. BLAINE RHOBOTHAM, D.D.S., Chicago, Ill.

ONLY a few generations ago, a very simple economic system prevailed. Farmers were still trading wheat, eggs and butter for shoes, dry goods and staple groceries; manufactured things were what the name implied — "hand made," and means of transportation and communication were slow and uninvolved.

Changes in our social and economic fiber have occurred with such phenomenal rapidity that we are almost lost in our attempt to adjust ourselves to these new situations. As a bloc example, study the predicament in which the American farmer finds himself today.

The industrialist, the agriculturist, even the great public itself, is looking for a helpful solution of the distressing condition that confronts the feeders of us all.

With the world as our market, this, the richest of all nations, is striving with untamed and unlimited energy to adjust itself to its own economic needs and responsibilities. Never in the history of man has there been such evidence of wealth as is seen in America today, and with it comes concern for the physical, social and moral welfare of the people.

The professional group is caught in this maelstrom of emotional and social readjustment. Naturally, there has arisen an acute interest in economic problems, and an attempt is being made to face them squarely and intelligently. Everywhere throughout the country, dental programs are including papers on dental economics, and study clubs are taking up this subject with evident interest.

When the necessity for an investigation of such a vital issue as dental economics arises, it should be met carefully and should be treated in the wholesome light of common sense.

Economics is a study of the material means of satisfying human desires. My endeavor in this paper is to show how a dental education as an investment may be analyzed and compared with other investments in material means to satisfy human desires.

An investment is the laying out of money for purchase of property, for income or for profit. It is compared with speculation in the old saying: "Planting good seed in fertile soil is an investment; betting on how many potatoes the seed will produce to a hill is speculation."

How great a material investment has the average dental practitioner made? Opinions differ greatly. For a working basis, let us say that the cash outlay and the cash represented by time spent in school, which might have been spent in profitable pursuits, amounts to $10,000.

Our first consideration beyond the actual money involved must be in the

*Read before the Illinois State Dental Society, Decatur, Ill., May 13, 1930.

*The papers of Drs. Rhobotham, Dittmar and Logan were presented as a symposium on "Dental Economics."

property in which it is invested. The "property" in our case is the individual. On careful analysis, do we find in the individual professional possibilities that may be developed by a dental education?

For the sake of further comparison, we will take it for granted that the individual is suited for the investment. The next study of the purchase lies in the wisdom with which the commodity is bought. Are we getting all that we are paying for? A wise buyer will take every fair advantage in obtaining his goods. That means that, in a dental training, the wise investor will get every valuable bit of knowledge that will sell advantageously.

To carry out the analogy, we will now assume that the dental graduate has purchased all of the dental equipment in his professional training that is purchasable for the price involved. At this point, the investment from an educational point of view has been made. His next hope is that this investment will do for him what all good investments should do—pay him an income. It is my hope that I may be able to suggest in what follows some of the means that may be employed to that end.

Why is it that some prosper in dentistry, while the great majority just get along? J. Ogden Armour once gave me a booklet entitled "That Something." It was a treatise setting forth the idea that some men possess "that something" which for them spells success.

Thoughtful of him to give a struggling young man that inspirational booklet! At that time, he was worth $250,000,000 and quite naturally he assumed he was one of those that possessed "that something" which seems to come only to a few. A fine man was J. Ogden Armour, but within a few years after his kind gift, he died in debt. Did he, after all, have "that something"?

We are told by some overzealous dental economists that "that something" is within the reach of all of us, and that if a definite system is put into practice, we may all become financially well off. I do not mean to minimze the effort of any man or any group of men that tries to help the dentist to a better understanding of his economic problems, but I do object to the notion that it is possible to work out a "system" which will insure a successful professional practice. A sound business system may be a means of general improvement to almost any neglected practice, but a universal business system offered to a profession from the outside is likely to commercialize the dentist to the point of neglect of our most sacred duties, which are service and mercy.

We need and should welcome outside aid, but primarily our remedies should come from within our own ranks, so that professional treatment shall always be our paramount consideration.

There are no tricks nor secret knowledge that need be sold to us to improve our financial income in our dental investment. Let us look into the offices of our successful confrères to see what they are doing that we are not doing. Do we find strange practices and foreign policies that we cannot apply? Certainly not; we find only sound, old-fashioned, common-sense ideas, carefully followed out.

Let us outline the sort of management one of these successful men employs to build up and maintain a lucrative practice. Let us step into his reception room and get a first-hand impression of a man who has made his dental investment pay.

Once inside this successful man's office, we are immediately impressed with the genius of the proprietor in his attempt to

convey a welcome to the newcomer and an air of congeniality to the old patient.

The reception room is, first of all, orderly, well furnished and free from an accumulation of last year's magazines. It is neither ornate nor severely plain, but it conveys an impression of the man within.

The door leading into the inner office opens soon after our arrival, and a neat, well-mannered assistant acknowledges our presence. Her graciousness and amiability at once put us at ease and we feel that our comfort is the prime consideration here. Our appointment has received businesslike attention and the office routine is so well managed that when it is time for our meeting with the dentist, we are ushered into the operating room.

What a feeling of reassurance we experience when we find every appointment immaculate and arranged so as to minimize any fear that we may have entertained. We are introduced to the doctor, who certainly fits into his surroundings. He is the personification of neatness, and, although his bearing instils immediate confidence, he is not austere; rather is he mild and sympathetic. There is no finer quality for a professional man to cultivate than that of sympathy.

It is our first meeting with our professional adviser, and he listens courteously as we explain our desire in seeking his services. He hears our inquiries patiently, and then explains that it will be necessary to make a complete examination before he will be able to give us intelligent advice. His assistant carefully marks a record chart, as he painstakingly goes over our mouth.

First, he examines the soft tissues, then the gross arrangement and the occlusion of the teeth. The supporting tissues of each tooth are checked, and finally every surface of the teeth is carefully examined and a record made. Then a general history is taken of the physical condition, temperament and all the other items that may concern the particular case.

The dentist explains the necessity for a full mouth roentgenogram that he may have all of the data before entering on a rational diagnosis and prognosis of the case. We have placed ourselves in his hands and we are impressed with his desire to proceed only after he has reached a complete understanding of the case.

He now gives us a notion as to the manner in which he manages the business side of his practice by determining before the next appointment is made whether or not his terms are agreeable. In a general way, he gives us a definite idea as to his fees for various types of service, and he explains that after making his final diagnosis from the roentgenogram, he will be able to determine within a reasonable amount just what we will be expected to pay. He further explains that he establishes credit according to accepted business methods and that the time of payments must conform to those rules. Could any arrangement be fairer? Having been further impressed by this honest, straightforward method of dealing, we decide to entrust ourselves to his care.

Before we leave, we remark that we are on our way to a ball game, and the dentist immediately gives evidence of his psychologic insight by showing familiarity with the game. The dentist must be well posted in order to show interest and acquaintance with whatever happens to be the patient's hobby and so reach his human side.

We are turned over to the assistant, who escorts us to the business office. There, we make an appointment, taking time to observe the orderly arrangement of desks and files. There is a dressing

room off the business office, which is put at our disposal before we leave. A cordial goodby from the nurse sees us pleasantly on our way, and we have left behind the office of a successful man. We are certain of several things: first, that we have seen every physical evidence of a prosperous dental office; secondly, that every incident of our personal contact gave reason for that success. So far, we see no tricks or strange practices to explain why he has prospered.

Suppose that we were so impressed by this successful dentist that we wanted to know more of him than we could learn merely by coming to his office for professional service: We cultivate him socially in order that in private conversation we may learn from him how he makes his investment in a dental education pay.

Seated comfortably at the club with this man whom we wish to emulate, we ask him what further he can tell us regarding his professional investment; and this is what he says:

"There are many elements to an investment but there are three outstanding ones that must be reckoned with always. The first is safety, which means that the investment should be made in securities that are stable and will not diminish in value. The second is liquidation, which means that, in case we wish to dispose of the investment, it can be sold readily at par value. The third is income. What return will the investment pay? A good investment must therefore be sound, must be liquid and must earn a fair return. Undue emphasis on any of these elements must be made at the expense of the other.

"How does this apply to investment in a dental education? The first element, safety, applies as follows: Before entering on the study of dentistry, one should be analyzed with these thoughts in mind: physical fitness, mental and mechanical adaptability and willingness to give of oneself unselfishly to a high calling of service to one's fellow man. Being passed on by an efficiency engineer or even by a psychoanalyst would be helpful.

"Willingness to enter into study wholeheartedly with the ambition to gain every opportunity offered by preparatory education and training is a further safeguard. During the time that must elapse before the investment has paid out, the money involved should be protected by sufficient insurance to cover the loss should disability or death terminate the possibility of earning returns. A further investment in postgraduate or research work must be accounted for, if the investment is to maintain its original value.

"Second is the element of liquidation. One's ability to transfer this 'property' quickly at its par value is the weakest point in the investment, for it can be disposed of only in part and never at par. Good will has a value far below par. Its greatest value in a transfer may be realized in gradually giving it over to a faithful co-worker or to a near relative, such as a son. In either case, the payment comes largely in satisfaction and in contentment, which in many instances have a value far above monetary expression.

"Third is income from the investment. This, the most important element in the final analysis of the scheme, depends on many factors. These may be listed as follows: Assuming that the investment has been wisely made according to the foregoing, it can be made to pay by the application of common-sense employment of the investment. Thoroughness, honesty, sympathy, mercy and service given courteously, tactfully, pleasantly scrupu-

lously and in good grace will meet the approval of the most discerning clientele.

"But all these finer qualities will not alone make for success. There must also be the application of approved business principles. These principles need not be complicated or confusing and should be included in dental school training. They are simply a careful record-keeping system from which costs, time accounts, discounts, disbursements and the like can be easily figured. The kind of system used does not make a great deal of difference, but one should be employed that is simplified and adequate. Collections and cost accounting can be systematized according to any of the approved methods so that they are simple procedures. If the dental school has not thoroughly prepared the graduate to understand these business systems, it is advisable to secure a simplified method through a study club or postgraduate courses."

We had expected to have our successful friend tell us some secrets of the inner workings of one who has arrived. But there are no secrets and we are where we started in our speculation on "that something" that spells success.

As a Hoosier would put it, it is simply work and the application of old-fashioned "horse sense."

55 East Washington Street.

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# THE TRAINING OF DENTAL STUDENTS AND YOUNG PRACTITIONERS IN DENTAL ECONOMICS

By G. WALTER DITTMAR, D.D.S., Chicago, Ill.

THE motive which evidently suggested "The Training of Dental Students and Young Practitioners in Dental Economics" for discussion is the charge which is frequently heard, that dental colleges have in the past neglected to give their students a course in what is now termed dental economics, and because of the lack of such training a large percentage of dentists have failed to be as successful in the conduct of their practices as they would have been had they received such a course. In this charge, it is plainly evident that financial success or business success based on commercialism is the primary thought.

If the obtaining of "the material means of satisfying human desires" (Webster's definition of economics) is the most fundamental thing which a prospective graduate of dentistry aspires to and craves, it must be conceded that the dental colleges have in the past failed to perform their full duty to such students in failing to teach methods which might be employed to increase the material means for satisfying human desires. And if the satisfying of human desires by material means is what is most desired by some practitioners in our profession, these practitioners soon make it evident that they belong to that class in the profession which is practicing for the money

*Read before the Illinois State Dental Society, Decatur, Ill., May 13, 1930.

it can make out of the business of dentistry.

It is true that dentistry, as a profession, like all other professions in this day and age, has definite business and economic problems, and it is well that they are being considered and studied by the profession; but these problems must ever be kept secondary to the true professional problems and must not displace the altruistic and ethical foundation upon which the professions of medicine and dentistry were built.

Dentistry is much more than a business, and the measure of a professional man's real success depends, in my opinion, on what was so eloquently stated by the late revered C. Edmund Kells when he discussed the question, "What Sort of Success Do We Desire?" Dr. Kells said:

> The man whose sole aim in life is a big fee, a large income, a big bank account, is a miserable failure in a profession devoted to the healing art. The practitioner who cannot take time to attend dental meetings, take part in the proceedings of his local, state or national society, assist his professional brothers or give attention to research work or teaching unless he sees financial gain, who is wrapped up in the scramble and greed for his own advancement, is an unworthy member of the profession. The man who takes no time to keep his body healthy, his mind broad, and his disposition sweet, is a flat failure no matter how much of a material success he may be. I wish to lay particular stress on the growing tendency on the part of some dentists toward the commercial attitude. This must be repudiated by the profession before it undermines the true ideals of our calling. What are these ideals? They are exemplified in the lives of the outstanding characters in our profession, many of whom have died in the harness, yes, shabby harness; but their memory lingers with us, indelibly written on the pages of history of our profession. They have given unstintingly of their accomplishments and substance for the betterment of mankind and have reaped the glory of service. We must not forget that we belong to this group, devoted to the art of healing.
>
> Success is within the grasp of every man, even to the most menial or humble of us, but the point of view must not be warped by the dollar mark.

It was men of the latter type, alluded to by Dr. Kells, who were the organizers of our first dental colleges—men whose fundamental training was grounded in the ethics propounded by the father of Greek medicine, Hippocrates, in the oath which every true physician honors and respects:

PHYSICIAN'S OATH

> I swear by Apollo Physician, by Asclepias, by Health, by Panacea and by all the gods and goddesses, making them my witnesses, that I will carry out, according to my ability and judgment, this oath and this indenture. To hold my teacher in this art equal to my own parents; to make him partner in my livelihood; when he is in need of money to share mine with him; to consider his family as my own brothers, and to teach them this art, if they want to learn it, without fee or indenture; to impart precept, oral instruction and all other instruction to my own sons, the sons of my teacher and to indentured pupils who have taken the physician's oath but to no one else.
>
> I will use treatment to help the sick according to my ability and judgment, but never with a view to injury and wrong doing. Neither will I administer a poison to anybody when asked to do so, nor will I suggest such a course. Similarly, I will not give to a woman an instrument to cause abortion. But I will keep pure and holy both my life and my art. I will not use the knife, not even verily, on sufferers from stone, but I will give place to such as are craftsmen therein.
>
> Into whatsoever houses I enter, I will enter to help the sick, and I will abstain from all intentional wrong doing and harm, especially from abusing the bodies of man or woman, bond or free, and whatsoever I shall see or hear in the course of my profession, as well as outside my profession in my intercourse with men, if it be what should not be published abroad, I will never divulge, holding such things to be holy secrets.
>
> Now if I carry out this oath, and break it not, may I gain forever reputation among all men for my life and for my art; but if I

transgress it and forswear myself, may the opposite befall me.

It is indeed not difficult to realize the force of this sublime statement and its influence on every true physician, past and present, though it was written approximately four centuries before the beginning of the Christian era. Then came the influence of that greatest teacher and spiritual physician, Jesus Christ, who gave to humanity—to every race and to every class—a code of ethics to be applied not only to a profession or a professional man, but also to every human individual: "Do unto others as you would have them do unto you."

The combined influence of these great teachings coupled with an earnest desire to serve their fellow men, developed such great characters as Horace M. Hayden, Chapin A. Harris, James Taylor, Eleazar Parmly, Norman W. Kingsley, J. H. McQuillan, Wilbur F. Litch, C. N. Pierce, Charles J. Essig, Oliver Wendell Holmes, Jonathan Taft, Homer Judd, Truman W. Brophy, Edgar D. Swain, G. V. Black, Edwin T. Darby, S. H. Guilford, G. B. Snow, J. D. Patterson, and a host of others, now departed, who conceived and brought forth in America a new profession—the profession of dentistry. Most of these men were physicians as well as dentists, and nearly all of them were the organizers or first deans of the colleges whose faculties they headed.

It is therefore easy to realize that these pioneers in dental education were imbued with lofty ideals, which, fortunately, were early reflected in the ethical code of the dental profession.

These high minded men and the scores of great dentists and dental teachers to follow them, endeavored or are endeavoring to impress their students and the young practitioners in dentistry with fundamental "Golden Rule" ethics. Thus, it is evident that the discussion of pecuniary and commercial questions connected with the practice of dentistry was not looked on with favor by most dental faculties a few years ago.

The professions of medicine and dentistry have, however, always had within their membership two widely divergent classes: the class which seriously subscribes to a professional code of ethics based primarily on the oath of Hippocrates and the Golden Rule, and the other class, which utterly disregards professional ethics, being made up of self-centered, pecuniary and often dishonest individuals. The one class is termed ethical, the other unethical. The ethical class has an ever-active feeling of contempt for the selfish actions of its unethical brothers, and since the basic motives that influence the unethical practitioners are, in the eyes of the ethical members, sordid and commercial, anything that was connected with the activities of the unethical class has been frowned on by the ethical members. To a great extent this attitude still exists, especially among college teachers, and has its influence in keeping out of some dental college catalogs the published announcement of a course of instruction in dental economics.

Modern ideas pertaining to economics were first outlined by Adam Smith in 1776 in his "Inquiry into the Nature and Causes of the Wealth of Nations." It is intensely interesting to note the marked changes in the status of economic conditions in the eighteenth century, especially in Europe, which were paralleled by the great "Industrial Revolution." The historian Dietz in his "Political and Social History of England" points out that the staying of the great plague in the seventeenth century; the discovery, in 1740,

of vaccination as a preventive against smallpox; the adoption of sanitary measures resulting in greater cleanliness, better homes, more abundant and purer water supply in cities, and a more detailed knowledge of human anatomy, curbed devastating diseases, lengthened the span of human life and consequently increased population in astonishing proportions.

Industry, which had for the most part been agricultural in form, now took on new forms; inventions of all kinds developed; transportation facilities improved; the manufacture of commodities increased; organization was made the keynote, first of capital, then of labor. Though organization from a military, ecclesiastic and political standpoint had dated back thousands of years, the industrial revolution, with its resultant economic changes, stimulated the organization of all forms of human endeavor, among them the learned professions. Thus, an organized dental profession developed in the early part of the nineteenth century, and into it as into all other forms of human activity, there has been and is now being forced the element that is spurring the civilized world to the utmost: commercialism.

In the past decade or two the greater part of the ethical element in the dental profession has been brought to a realization that economic problems, big business and commercialism affect the manner of conducting a dental practice today in a more marked degree than they did even a quarter of a century ago. These problems have similarly influenced our dental publications, our big dental meetings and our dental colleges. It is interesting to note in the 1929-1930 catalogs or bulletins of thirty-two of the leading dental colleges in the United States and Canada (all that I could procure a copy of and carefully check) that twenty-eight announced a course in dental economics. Thus 88 per cent of the curriculums examined announced economics as a subject taught to the senior class. Practically all gave courses in jurisprudence and ethics, and a large proportion, in dental history. Under the heading, "economics," some of the bulletins gave a brief statement as to the content of the course; for instance, "The business requirements for professional success in dentistry" — sixteen lectures to the senior class, or "A study of overhead costs together with a consideration of educational investment is strongly stressed in order that a fair and remunerative compensation will result from the practice of dentistry." Some of the catalogs gave a much more comprehensive announcement, stating that the course consisted of thirty-two lectures to the senior class, including class exercises, bookkeeping, etc. The following is a fair example:

ECONOMICS

Consists of lectures given with the intent to reveal the business problems of a dental practice to the student before graduation, in order that he may successfully meet the obligations assumed in a manner that is honorable. The lectures cover the following topics: selection of a suitable location; office arrangement and equipment; office records; reception and business relations between dentist and patient; care of credits; collections and payment of bills; office management, buying of materials; fees; banking; insurance and investments.

Ethical publicity and such personal factors as cleanliness, neatness, reliability and punctuality, personal efficiency and the psychology of professional success are given in such courses.

A list of the names of books, reports and essays and their authors to which the student is referred for study is also of interest: "Wealth of Nations," by Adam

Smith, "Human Efficiency," by H. W. Dresser, "Efficiency," by H. Emerson, "Conduct of Life," by Ralph Waldo Emerson, "The Efficient Life," by L. H. Gulick, "Success in Dentistry," by L. M. Hendrick, "Talks on Psychology and Life's Ideals," by William James, "Psychology and Industrial Efficiency," by H. Musterberg, "Business, a Profession," by L. D. Brandies, "Essentials of Business Law," by F. M. Burdick, "Past and Present:" Two essays: "Captains of Industry" and "Plugson of Undershot," by Thomas Carlyle, "Accounts, Their Construction and Interpretation," by W. M. Cole, "Elements of Economics," by R. T. Ely, "Success in Dental Practice," by C. N. Johnson, "Ethics and Jurisprudence," by Edmund Noyes, "The Dentist's Own Book," by C. Edmund Kells, "History of Dental Surgery," by C. R. E. Koch, "Dental Jurisprudence," by Elmer D. Brothers, "Munera Pulveris: Six Essays on the Elements of Political Economy," by John D. Ruskin, "Increasing Human Efficiency in Business," by Walter Dill Scott, "Dental Education in the United States and Canada" (Bulletin No. 19), by William J. Gies, "Theory of Business Enterprise," by T. Viblen, "Economic Factors in the Management of a Dental Practice," (THE JOURNAL, February, 1929), by Charles R. Lawrence, "Dental Economics," (THE JOURNAL, May, 1930), by Harold J. Noyes. In addition to these, there are many instructive and interesting editorials, essays and surveys on dental economics appearing regularly in the numerous dental periodicals.

From the foregoing, it is evident that there has been much written on the vital subject of economics, and that a large percentage of the dental colleges do train their students in the fundamentals of this science. If dental economics is taught in connection with the history of dentistry, dental ethics and dental jurisprudence, as is done in most colleges, their students will be well grounded in the truly professional and ethical phases of the practice of dentistry, and thereby be safeguarded against the purely commercial type of unprofessional teaching.

With reference to training the young practitioner in dental economics, I will quote a brief extract from an editorial[1] by the dean of Tufts College Dental School, in which a valuable suggestion is made:

> Much has been said and much written of late on the subject of dental economics. Why the term dental economics? Economics has been defined as the science of wealth or the material means of satisfying human desires. Economics as taught today in universities throughout the United States and other countries is as directly applicable to the dental problem as it is to general business interests. Why encumber further the dental curriculum already overloaded with extraneous subjects? Is there any reason why the practicing dentist or dental student should not take advantage of the summer extension course in Economics given in all of the larger universities? The usual course is of six weeks' duration; twelve hours of instruction are given each week. The average cost is forty dollars. As a matter of fact the dental student may include economics in his predental study. The dental profession seems to be oblivious of this opportunity. The dentists are flocking to the one-man courses, for which they are paying a comparatively large fee and getting what many consider questionable advice as to ways and means of increasing their income.

These certainly are excellent suggestions for the practitioner both young and old and for the dental student, since the subject of general economics is fundamental to all of the special branches thereof.

Reference to the "one-man" courses to

1. Rice, William: Dental Economics — A Possible Solution of the Problem, Bull. Massachusetts Dent. Soc., Dec. 30, 1929.

which many dentists are being attracted, and for which, seemingly, they gladly pay large fees to get what many consider and what Dr. Rice terms, "questionable advice" is worthy of special consideration. Some of these courses may be undesirable from a truly professional standpoint, but like most objectionable things, they finally provoke counteraction or at least lead us to seek a remedy. So has it been here. These "one-man" or commercial courses have aroused a desire on the part of the ethical element in our profession to study the economic question from a fundamental as well as from the special dental aspect, and at the present moment, as has been previously stated, the dental colleges, the dental journals and the dental societies have responded to this feeling by earnestly studying this subject.

The young practitioner therefore has abundant opportunity to gather from the writings presented on the subject of economics or from a course in economics at some university such fundamental material as will apply to the particular location that he may choose, or has chosen, in which to conduct a dental practice. He should, of course, so plan and organize the business and economic phases of his practice that he will be successful in obtaining an adequate amount of the "material means for satisfying human desires," and judiciously save and invest enough of it to be enabled to care for his loved ones and himself in the event of a "rainy day" or during the evening of life. It is sad indeed to see the examples so frequently brought to our notice of capable, earnest, conscientious dentists who have given of their best efforts for many years to their patients and to the uplift of the profession, yet who because of some misfortune, but more frequently the unfortunate lack of applying sound economic principles in either conducting their practice or in making investments, or both, have saved nothing and become a charge to their loved ones, or to the profession or the state.

The study of economics, in my opinion, should have a place in the curriculum of every dental college, and it should likewise be studied and practiced by every dental practitioner; but by no means should the student or practitioner ever permit the greed for the dollar or the principles of trades unionism to supplant the sublime ethics of the Physician's Oath or the Golden Rule. Dentistry is much more than a trade or a business: it is a noble profession, and it is our duty to preserve its honored traditions.

# THE TRAINING OF DENTAL STUDENTS AND YOUNG PRACTITIONERS IN DENTAL ECONOMICS*

By WILLIAM H. G. LOGAN, M.D., D.D.S., Chicago, Ill.

BY agreement with my colleagues in this symposium, I shall confine my remarks to that phase of dental economics which deals with the first consultation the patient has with the dental practitioner, at which time a general history is taken and notations made as to past and subsequent services to be rendered. Naturally, you may wonder what relationship exists between the initial consultation and the subject of dental economics. Perhaps this can be clarified by my interpretation of the definition of dental economics as it relates to the particular phase of the subject that I am to discuss. Dental economics is that science which deals with all the factors that enter into the rendering of a professional service for an appropriate fee. It is this problem in dental economics in which I am particularly interested, for surely no one should be concerned about the receiving of larger fees if better service is not rendered.

At the present time, the majority of professional men, regardless of whether they are academic or professional teachers, or practitioners in a village, town or city, seem to be in need of an increased net income. As a substantiation for this recommendation, it is well to remind you that, in the majority of instances, out of every thousand dollars received by the dentist, not more than $500 to $650 can be looked on as net income. In a recent survey[1] of medical costs to the public, which included dental service, it was ascertained that 22 cents out of every dollar is paid to the physician, while 17¼ cents is received by the dentist. An item that must be taken into consideration is that the upkeep of the dentist's office and his expenses for materials are in excess of those of the physician; while his professional expenses outside his office are materially less. On the other hand, the physician is required to spend more years in preparation for his life's work.

Although it is affirmed that failure overtakes nine out of every ten who engage in business for themselves, and that the percentage of success among professional men is far greater than among those who enter into trade, it seems to me that there is much to be gained by a plain and thorough discussion of the question of dental economics. Furthermore, I do not fear a frank presentation of this subject as do some who contend that such an agitation may result in the establishment of free dental clinics, or that a vast number of clinics may be organized by the wealthy for the poor or middle-class pa-

*Read before the Illinois State Dental Society, Decatur, Ill., May 13, 1930.

1. Davis, M. M.: Doctors' Bills and People's Billions, J. A. M. A. 94: 1014 (March 29) 1930. (The estimate of 17¼ cents out of each dollar being paid to the dental profession is computed on the basis that there are about 2¼ times as many physicians in the United States as there are dentists.)

tient, and that such clinics may be managed on the cost basis. The dental profession has little to worry about in discussing its financial problems, for if there is something wrong here, the more promptly it is righted, the brighter the future will be, not only for the public but also for those in our calling, provided we approach the subject with the declaration that dental economics deals with the rendering of professional service for a just remuneration.

From my own experience, I am led to believe that the members of the dental profession are not quite clear in their own minds as to just what they are entitled to in the way of compensation. Although I cannot answer this question, permit me to state that I believe that the average professional man engaged in law, medicine or dentistry, is justified in making a laudable effort to secure for himself and those dependent on him such comforts, pleasures and educational opportunities as are enjoyed by the average of the group that he serves. If a dentist, as a result of his educational qualifications, his professional ability and his value as a citizen to the community, has attracted a given group of patients who occupy his time rather fully and whom he serves faithfully, he should expect to have as many of the comforts and pleasures of life and educational opportunities as the composite of the group that he serves, and in the autumn of a full life of worthy service, he should be as financially independent as the average of his patients.

Without going into detail, I ask you: Is there any justification for the average dentist to feel that he should be as financially successful as the wealthiest of his patients? Yet he should not be satisfied with the lot of those in the lower strata of society; for is it not true that, in the long run, men ascend or descend to the plane they are willing to accept for themselves, except in those instances in which sickness or other calamities overtake the practitioner or his family.

With the two foregoing fundamental statements in mind, that there is a definite dental economic relationship between the professional service rendered and the fee to be received, and, secondly, that the practitioner should at least aspire to the status of the average of those he serves, we are now confronted with the necessity of dealing with ways and means that will assure the practitioner the attainment of that which we claim is his just due. My approach will be made through an effort to reply to the question, "What has the average patient, in return for the fee you are going to demand of him, a right to expect when he first comes in for consultation?" To state it another way: What must you do to increase the confidence of the person coming in for the first interview to make of that individual a satisfied and permanent patient?

In dealing with this question, we must be very explicit if the best interests of the patient are to be served. Our obligation is to ascertain all the factors that enter into a diagnosis, and this can be reached only by including other structures than the exposed surfaces of teeth, yet these too must receive our careful inspection at the appropriate time. One of the grave mistakes that our profession has never corrected is that we have continued to care for our patients in the same manner that our associates in medicine pursued until perhaps half a century ago; i. e., when a patient came in, the physician inquired regarding his symptoms and treated him accordingly, without making an investigation regarding the health of the entire body. This resulted in failure to discover many pathologic conditions in their early stages of development. It

is not uncommon even now, in the majority of dental offices in this or any other state, when patients come in for the first time, for the dentist merely to examine the teeth for cavities or for spaces from which teeth have been previously extracted, and then proceed with his restorative operations, even though such preliminary work as the filling of root canals may be necessary; or crowns and bridges are sometimes placed without information first being sought as to the general health of the patient, or without note being taken of abnormal conditions in the mucous membrane of the mouth and underlying tissues, and the absence or presence of diseased areas of the jaws or root fragments over which he may be placing dentures or bridges.

I am not alluding to this known weakness on the part of our members simply to call attention to the fact that they are overlooking an item that has a great influence in their economic success, but rather to stress the importance of the definition of dental economics as outlined at the beginning of this paper; for dental economics is that science which deals with all the factors that enter into the rendering of a professional service for an appropriate fee. These statements do not seem to me to be far afield in a discussion of this character.

Although in the Chicago College of Dental Surgery, Dental Department of Loyola University, dental economics and ethics have been taught by special teachers for a continuous period of eighteen years, I doubt that a single year passes that I am not asked by some graduate why he did not receive instruction in this very subject, I knowing full well that this subject was included in his course of instruction. With this in mind, we have from time to time included in our clinical instruction an increased number of requirements that we feel will be helpful to the students from an economic standpoint when they become practitioners. For example, in the early days, a patient coming to our dental clinic was not charged a distinct fee for the cleaning of his teeth, but later a charge was made for this service, in order to impress on both the student and the patient that it was a distinctive service worthy of compensation. Likewise, a single fee was charged for all root-canal fillings and the placing of crowns over them; but this was changed so that a separate fee was charged for each operation, although the total charge was not increased. Later, a careful charting of all teeth and restorative work then found and needed in a given mouth was demanded before a student could begin to operate, and for the purpose of developing his diagnostic ability, he was called on to make his verbal recommendations to an instructor and he was also urged to secure the approval of the patient as to his conclusions.

Although this was a great improvement, we felt that both the student and the patient were in need of additional training for the best interests of all concerned. So, we have recently established a new division of instruction which seems to us to have an important bearing on the proper development and point of view of both the oncoming young practitioner and the patient with regard to the entire question of dental economics.

This new division has been designated as the Department of Diagnosis, but it could perhaps more appropriately be termed the Department of Clinical Diagnosis and Dental Economics, for here the patient is given an opportunity to become acquainted with all his oral and dental needs, and is called on to pay an appropriate fee for the service rendered. When the patient comes to the

dental clinic for the first consultation, a dental student records on a diagnostic chart before him a complete outline of the condition of his mouth, teeth and supporting structures. In the presence of the patient and the student, these findings are then reviewed by competent full-time teachers who have no other duties. We require that all patients pay a very nominal fee for this initial examination, and all patients coming in for restorative work at the present time are required to submit annually to a complete oral and dental examination; and these findings are recorded on permanent history charts, which are filed and kept for comparison and reference in connection with the subsequent annual examinations. For such annual reexaminations, a distinct nominal fee is required, the purpose being to impress on both the student and the patient that such a procedure is the only safe one to pursue if the dental health of the public is to be safeguarded, and the dentist is to be treated fairly from an economic point of view. It is estimated that the present consultation fee paid by the patient in our dental clinic covers between 30 and 40 per cent of the actual annual expenses; yet we feel that the purpose for which it was instituted is being served.

The consultation procedure, as we see it, should be one that will completely portray at a glance all restorative work to be found in the patient's mouth, as well as the indicated operation required, for this will permit the patient to better appreciate the extent and difficulties that must be overcome. If the original record and the subsequent ones reveal the essential facts, the dentist, if he has faith in his ability, will be able to decide what would be a fair fee for the services rendered for the various operations.

I am satisfied that the misunderstandings that sometimes arise over fees, or the lack of courage on the part of the practitioner to demand that which is his just due, would be changed if the patient clearly understood his dental needs, for no reasonable patient desires service to be rendered at a loss. No—the difficulty lies in the dentist's lack of faith in himself and his unwillingness to so apply his time that he may be conversant with the factors that would lead him to professional and financial success.

I now invite your attention to a plan which I believe to be one important phase of the proper teaching of dental economics to junior and senior students in the diagnostic clinic of the institution that I represent. But I also wish to say to those of you who graduated from any dental school in the years that have just or have long since passed, that this procedure can be applied to your own practice for your professional and financial advantage, and for the betterment of those who seek your advice.

I wish again to emphasize the point that we favor making a complete written report of the findings regarding the oral cavity and the teeth and their supporting structures, and then reviewing annually this original history, recording thereon such variations as appear from year to year. Furthermore, the plea is made that since a valuable service is rendered by such annual examination, the patient should be called on to compensate the practitioner with an appropriate fee.

Determination of the fee to be charged for the annual examination or subsequent professional service must naturally depend on overhead expense and the ability of the patient to pay. Since the dental institutions which graduate the students cannot be made aware of these factors, the responsibility for establishing ap-

propriate minimum fees for the different types of operations must be left to the practitioners in the localities in which a recent graduate locates. If the local organized profession fails to make this information available to the new professional resident, they have not accepted the responsibility that rightfully belongs to them. It is impossible for a teacher of dental economics to inform a student as to what the minimum fee should be for various types of operations unless he knows under what circumstances the work is to be rendered. If there are those among you who have this information and the belief that it will stand the test of practical experience, we welcome your advice; but until you are able to advise each other as to what the appropriate fee should be, I believe it is unfair to criticize the teaching institutions of this country for not imparting this knowledge when they do not know the circumstances surrounding the future practice of their students.

Before dismissing this phase of the question, I desire to express the opinion that students and young practitioners should be informed that there is a rather close relationship between the expense of maintaining an office and the fees demanded, and that relationship should be on the basis of one-third for actual expenses and a minimum of two-thirds net for the practitioner; and I believe that conditions should be so changed that the net would equal three-fourths of the gross income.

Having affirmed that practitioners can wisely consider the establishment of a minimum fee for a given locality when service is rendered under like conditions for the average patient, I wish to say that I am also of the opinion that there should be a maximum fee even for those most able to meet their financial obligations, and this maximum fee might appropriately be placed at not more than ten times the minimum fee, unless there is something very unusual associated with the case.

Probably, at the present time, the greatest need of the dental profession is not a new system for the purpose of demanding excessive fees from patients or for additional knowledge with regard to technic, but a reconsecration of professional ideals, a dissemination of the facts of the relationship which exists between the gross and net income of the dentists and a closer application to the needs of our patients so that they may never leave our offices without being informed as to just what is best for their dental health.

Likewise, the quickest way to increase one's professional income is not to master lessons in the technic of high pressure salesmanship, but rather to take an audit of one's weaknesses as revealed in those characteristics which go to make up personality, and then set for one's self the task of improvement through observation and self-education by the dedication of at least a few hours a week to the reading of cultural and professional subjects. Thus the professional man may become thoroughly conversant not only with city and national events, but also with international affairs. In this way, he will make of himself of influence in his community as well as in his profession. He should never forget that he is entitled to a just remuneration for any honest service that he renders, and he should thereby develop the courage to demand it so that those dependent on him may have the comforts that rightfully belong to sincere professional men.

I desire to close this presentation in the same manner as I opened it—with my definition of dental economics: Dental economics is that science which deals with

all the factors that enter into the rendering of a professional service for an appropriate fee.

[In the presentation of his paper, Dr. Logan showed slides to illustrate the diagnostic chart, with the daily and hourly record sheet of all operations performed by the students in the Chicago College of Dental Surgery, by means of which every school hour of each boy's time is accurately accounted for. It is also a constant reminder of the necessity of keeping records and the most approved plan of estimating the proper compensation for all kinds of operations.]

# PARTIAL DENTURES: TYPES OF ATTACHMENTS AND INDICATIONS FOR EACH*

By ROY JAMES RINEHART, D.D.S., F.A.C.D., Kansas City, Mo.

A GOOD-SIZED volume might be written on the subject of "Attachments and Indications for Each," but if it were simmered down to a personal point of view, a few paragraphs might suffice. Most of us try all new ideas, and it seems to be the experience of the majority that just about the time a problem seems to be solved and things are looking fairly well, reports of failures and shortcomings appear, and again we realize that most operations in dentistry are more or less temporary. We have found that discarding old things for new is not always the act of wisdom, and we are compelled to return to those things which have proved quite successful. It is a noticeable fact that the profession as a whole is ever adopting better technical procedures. We are constantly looking for the ideal, so if we are compelled to return to some of the older methods, we will at least return with the idea of improving them.

Dental literature reveals to us that clasp attachments have been used as far back as we have records of teeth being supplied. We will first take into consideration what constitutes the difference between a removable bridge and a partial denture. The partial denture comes under the classification of tissue bearing cases, while a removable bridge is dependent for support on abutments that are clasped. As a rule, most removable appliances are a combination of the two forms of support. Norman B. Nesbett, of Boston, states that he designed his cast clasp as a stationary or rigid support. His clasps were stationary and nonflexible and took the entire strain of mastication. His bridge was anchored at both ends. Many operators adopted his clasp as a means of retaining partial dentures with saddles resting upon the tissue. These clasps, being nonflexible, soon brought disaster to the teeth on which they were placed. Modifications of this clasp have been many. Among those prominent in this work today are Kennedy, Roach, Akers, Cummer and Chayes. All have contributed valuable ideas, but no system devised by any one of these men will solve the

*Read before the Section on Partial Dentures at the Midwinter Clinic of the Chicago Dental Society, Jan. 14, 1930.

problem for the conscientious and intelligent operator.

Each case presents a problem of its own, and determination as to just what kind of attachment shall be used in an individual case demands careful thought and plans for the procedure. Cummer and Kennedy have probably given the profession the most comprehensive method of classification. Bailyn of Hartford, Conn., in a paper read before the Connecticut State Association, gave what he terms a more simple method of classification. With a comprehensive system of classification will come a better understanding of the principles of partial dentures as well as the attachments to be used. Let us first classify partial dentures and removable bridges in three groups: (1) tooth-bearing cases; (2) tissue-bearing cases, and (3) a combination of tooth-bearing and tissue-bearing cases.

A tooth-bearing case is one with anterior and posterior attachments, the attachments being constructed so that they will rest at a definite place on the tooth. In the case of the clasp, the occlusal rest acts as a stop for the removable bridge, maintaining it in the position for which it was constructed, regardless of the tissues under the saddle, if a saddle is used.

Tissue-bearing cases are strictly partial dentures or plates. The slot or tongue and groove attachments, of which there are several designs, and clasps without rests are often used in case of tissue-bearing dentures. The case is allowed to settle at all times with the tissues.

Tooth-bearing plus tissue-bearing cases are those in which the clasp or any form of attachment does not allow the saddles to settle completely upon the tissue, but holds the occluding surfaces fairly well in place.

## DIAGNOSIS AND DESIGN FOR RESTORATION

A thorough diagnosis of the case is most important. We will give three steps in diagnosis: (1) a thorough clinical survey of the entire mouth, including tissue examination, and determining the flexibility of the mucosa and pathologic conditions, if any; (2) a roentgen-ray examination of the entire mouth to determine the hidden conditions of the roots of the teeth, involvements of the pericementum or periapical conditions, and the firmness and health of the alveolar process, etc.; (3) the making of study casts, for a technical survey of the relation of the teeth, spaces, tooth form and occlusion. These are also very necessary when making a decision regarding the teeth to be clasped, type of clasp, saddles, etc.

An intelligent builder of a bridge or a structure of any material consequence would not think of proceeding without definite specifications. Occlusion of the teeth should be studied in detail so that the case under construction will have occlusal form corresponding to that of other teeth in the mouth and applicable to the movements of that particular case. We will list a number of the attachments which are being most successfully used: (1) cast clasps with occlusal rest; (2) wire clasp with and without occlusal rests; (3) half round wire clasp; (4) wide flat band clasp with and without occlusal rest; (5) bar or finger with clasp; (6) double bar stabilizer and clasps; (7) tongue and groove attachments.

## INDICATIONS

The most common indication for a removable bridge is the loss of a first molar. Thus, a typical bridge case is best constructed out of any of the specially prepared gold alloys for cast clasps. In preparing the teeth for this case, as in all

others in which clasps are used, the teeth should be thoroughly cleaned and polished. The only grinding is done to relieve the marginal ridge enough to receive an occlusal rest with sufficient bulk of gold alloy to stand the stress of mastication. While this preparation does not require the loss of much tooth structure, this loss must be greater than many operators have anticipated. This case is entirely tooth bearing. The clasps are ground and finished on the inner surface so that they present a three-point contact; namely, the occlusal rest and the two ends of the clasp. These points should be well polished, but not ground. The inside of the clasp is ground with a small round mounted stone so that it is entirely relieved gingivally, sloping to the occlusal margin. The occlusal surface of the tooth which is being supplied should be spot ground until there is no cusp interference whatever. This is true with all kinds of removable work, and I am convinced that this idea should prevail with fixed bridgework and full denture construction as well. Digressing just one moment from attachments to occlusion, I am in accord with those who have declared themselves as being against employment of an occlusal form which causes undue stress on the appliance and abutment teeth during the process of mastication. The success of all substitutes is dependent largely on free movement of the occlusal surfaces without cusp interference.

The second class that we will consider is the space between the cuspid and the second or the third molar. The attachments used in this case may be either cast or wrought wire. A flat, broad bar should extend across the roof of the mouth at the junction of the hard and soft palate, the area which presents the least obstruction to the tongue. An extension of this bar is carried to the interdental space between the second bicuspid and the first molar on the side where no teeth are missing. Here, a double clasp is constructed after the marginal ridges of the bicuspid and the first molar have been ground for the reception of the occlusal rest. If a wire clasp is used on the molar of the opposite side, the cast clasp, gripping the tooth labiolingually, will be found more nearly permanent for the cuspid. If there is no molar, but all the teeth are missing from the cuspid back, on one side, the case may be constructed as the former with an indirect retainer extending from the region of the double clasp to the lingual aspect of the cuspid, or it may also extend to the mesial marginal ridge of the first bicuspid.

Care must be exercised at all times that indirect retainers do not act as orthodontic appliances, or, in other words, that, if the case should settle slightly, the indirect retainer does not push the tooth forward. This would be avoided in case the indirect retainer extended on the mesial aspect of the first bicuspid, including an occlusal rest. If the operator studies his case thoroughly, he will construct indirect retainers that keep the denture in perfect balance and will not move the teeth.

The next typical case that we will consider is a bilateral type, in which there is an absence of all of the teeth on both sides back of the first or second bicuspid. While this type of case can be handled quite successfully with broad saddles and a broad connecting piece across the anterior part of the mouth, using clasps or tongue and groove attachments and allowing the saddles to rest entirely upon the soft tissues, my experience and observation leads me to believe that the use of a skeleton design with cast clasps and occlusal rest or of a drawn wire type with occlusal rest, both being designed with

some form of indirect retainer, is a better method of procedure. The three-point type of cast clasp, which gives some movement and spring, is quite successful. A swaged clasp of 18-gage wire with occlusal rest attached to 16-gage wire used as a stressbreaker, one buccally and one lingually attached back on the saddle, is very useful. The same principles that I have outlined above are indicated in practically all cases in which there are missing teeth in from two to six areas in the arch.

A difficult case in the lower jaw is found when the six anterior teeth remain. Dr. Kennedy suggests, in addition to clasps, a double bar, one being across the lingual aspect of the teeth and acting as a stressbreaker, or perhaps we might call it a stabilizer. This case, constructed as Dr. Kennedy suggests, gives excellent service; yet I am sure that much care of these teeth is required to avoid decay. It is true that the teeth remaining may not be expected to last many years anyway, and if while they do last, the patient is comfortable and the power of mastication is appreciably increased by using this form of stressbreaker, we are justified in using it. An accurately constructed clasp of wire, using the swaged method, is very effective. Dr. Jordan, of Kansas City, has done much successful work with wire clasps. Originally, he made a wire basket clasp which was quite efficient. This clasp had some features of the Jackson crib clasp.

### TONGUE AND GROOVE ATTACHMENTS

Every type of attachment that has been successfully used by any operator has its place, but those that require an extreme technic for their use are not applicable to general practice. This is the first advantage of a clasp, either cast or made of wire, over the tongue and groove type.

The second advantage of the clasp is that it does not require excessive mutilation of tooth structure. The cast clasp that is made of a strong flexible alloy ground on the inner surface and thoroughly polished is probably used most extensively today. Whether it is to be used with a bar or finger projection or directly cast as part of the saddle at the contact point demands an intelligent decision of the operator. The tongue and groove type of attachments have failed to become popular. My observation is that most operators, including competent ones, have encountered too many failures. Most of my own personal cases needed an excessive amount of readjustment and finally a change to clasps, or to have a clasp added. I see no reason for excessive mutilation of perfectly sound teeth in the case of removable bridges. There might be a reason for it when the patient insists that no gold show and when there are already defective teeth to consider.

### CLASPS INJURIOUS TO THE ENAMEL

It is argued that clasps abrade the enamel and that the teeth may decay under the clasp. This was true with the old close-fitting clasp, but it is certainly not true with the present method of clasp construction; nor is it true where wire is used. My experience with cast clasps extends over a period of about fifteen years, and with varying results; but within the past two and one-half years, my personal contact and observation has extended to more than 400 cases, many being constructed with the modified cast clasp. There has been no evidence of decay and very few of them have returned because of breakage. These clasps have been attached both directly at the contact point and with finger projection.

The three-point contact clasp with variations and the wire clasp with occlu-

sal rests offer a quite satisfactory variation of practical attachments, as does also the use of indirect retainers and stress-breakers. Naturally, the patient must be instructed in the care of both the teeth and the denture. Cleanliness is the watchword, and the patient should be asked to return at any time that the denture is not perfectly comfortable.

### CORRECT DESIGN OF THE CLASP

Each clasp to be used in the case should be designed on the plaster cast and it must be designed to work in harmony with the other attachments. The contact point is the place where the clasp should begin. The clasp must extend gingivally just over the bell of the tooth. It must so perfectly grasp the tooth that the denture will not be misplaced under the stress of mastication. Each clasp in the case must be designed to be parallel with every other clasp on the case or the appliance will not go to place as indicated. While it may be to our advantage to have an expensive instrument that will design clasps, it is not an arduous task to use an "Eversharp" pencil with the lead extended quite a distance. When the cast is held in one hand and the operator looks straight down on the teeth, the location and form of each clasp can be definitely designated on the plaster with a pencil so that the clasps will all be parallel when finished. This procedure, which is almost indispensable, has a distinctive advantage over designing one clasp at a time on separate impressions.

### PATIENT EDUCATION

As a profession, we are interested in education of the laity regarding the importance of good dentistry. Our dignified advice and conduct should express the standards of our profession. The profession is at an important place in its journey. We believe in always giving credit where credit is due. If you give me an idea, professionally, I owe it to you to give you the credit. An exchange of ideas has meant progress in dentistry, but when these ideas are commercialized, it means a hindrance to progress. If the dentist in his office engages in the sale of a commercialized idea or system, he is not engaged in the practice of legitimate dentistry. The only thing a dentist has to offer is his professional service. No one idea or system meets the universal need. A dentist should have judgment, ability, knowledge and individuality enough to develop his own outline of practice; namely, an honest and adequate service consisting of the application of those things that best meet the problem of the individual case.

Very soon, if we do not watch, we will be just order takers, selling some particular manufacturer's teeth with which patients have become familiar through loose and unintelligent sales talk of the dentist. The dentist who depends on a laboratory for advice and consultation and recommends a stereotyped case has acknowledged his incompentency at the beginning, and his patients will surely find it out eventually. There are several types of partial dentures being offered for sale by name in many dental offices in the same manner in which you would sell an automobile. No doubt, advice is given the patient according to the service offered by the laboratory.

Then, there is Dr. Blank's system of orthodontia. All this is becoming a part of the patient's education, and some of these days, when all we have to do is to greet our patient pleasantly in a "high powered" office and listen to a diagnosis of his own case and accept an order for a certain brand of restoration, this "pa-

tient education" will place the profession of dentistry in an embarrassing position, and real dentistry as a health service will have a terrible setback. "Patient education" of this type is just one half the capricious story that leads us to the subject "What Will Constitute the Future Practice of the D.D.S.?"

# HISTORY AND PROGRESS OF THE CAST GOLD INLAY*

By FRANK H. VORHEES, D.D.S., Chicago, Ill.

NO one phase of modern dentistry has received more attention from our profession than the use of the cast gold inlay. The claims made by the various writers and technicians all over the country almost remind one of the stories of the ardent golfer and his score, or of the fisherman and his catch. At times, the battle has raged fiercely.

Inlays of porcelain and glass were made as early as 1820, and the technic for this method was fully described and recorded in a crude way. Gold inlays were first made as a result of the experience of an unfortunate gold foil operator who, in finishing and polishing a filling, dislodged it when the operation was almost completed, but, not to have his purpose thwarted, rescued the filling and cemented it to place with whatever cementing medium was in vogue in the early part of the eighteenth century. Perhaps, even at this late date, many in this audience can recall a few instances in their own offices, when they have slipped in making a gold foil filling with the tactics used in the early days. The name of the first unfortunate operator to employ this method of rescue will probably never be known, but it is safe to say that the procedure dates back as far as the beginning of the first work in gold foil.

In the "History of Dental Surgery," by the late C. R. E. Koch, gold foil for the filling of carious teeth was mentioned as being used as early as 1450. Next came the gold inlay made by burnishing a matrix of thin gold or platinum to the cavity surface and investing it and flowing gold solder in to fill the matrix. This method was used extensively by Drs. Ames and Swasey of this city as early as 1880. A perusal of the dental literature of the early periods reveals various methods for making gold inlays.

With all the activity of the early periods, it was not a popular procedure as a means of treating caries until 1907, when William H. Taggart introduced to the profession his method for the casting of a gold inlay.

There seems to be considerable difference of opinion as to the propriety of Dr. Taggart's claim for the originality of this method. I have made a careful search of the literature on this subject, and while perhaps Dr. Taggart may not have been the first man to make a gold inlay by the casting method, it was he who wrote on this technic and published it. He gave clinics from coast to coast and made it possible for our profession to take advantage of this most successful method of

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*The papers of Drs. Vorhees, Sundby, Goering and Meyer were presented as a symposium.

reproducing the wax pattern of a cavity in gold. Dr. Taggart's first paper and demonstration of his casting method was presented in January, 1907, before the New York Odontological Society. At this meeting, the key note was sounded, and a great step was taken in the interest of better operative dentistry.

The results achieved at this meeting caused one of the greatest ovations ever witnessed at a dental meeting. Yet, it is only reasonable to assume that only too few who were fortunate enough to attend the meeting could visualize the importance of Dr. Taggart's accomplishments. It not only marked the beginning of better operative dentistry, but also, as a result, the casting art was soon applied to the various phases of prosthetic dentistry with a degree of success that meant better service to the patient. It revolutionized dentistry, created higher standards of craftsmanship and was, no doubt, the beginning of a new era.

With the advent of the casting process as given by Dr. Taggart, it became necessary to revamp the crown and bridge department of every dental university in the world; for, with it, we were able to step to a higher plane of service than ever before. We came into possession of knowledge enabling us to do things of which we had never dreamed. Is it any wonder that we should take advantage of this golden opportunity to pay homage to this great man, the father of it all?

From 1907, for a period of almost twenty years, little, if any, scientific knowledge was added to Dr. Taggart's efforts. Few, if any, were able to surpass his efforts and only too few to equal them. Yet, in spite of the soundness and thoroughness of the principles laid down by him, there have been many hundreds of different ways evolved to make an inlay, but none with any greater success than that outlined by Dr. Taggart. Various attempts have been made to overcome the few shortcomings in the original technic, but it has taken almost twenty years of research to improve, in any appreciable manner, on the first basic principles laid down by Taggart in 1907.

The contraction of all inlay golds is greater than the coefficient of expansion of our present inlay investments. This difference was not a serious one when dealing with the small castings, but, in the three-quarter or full crown, it was a factor that has been a stumbling block and a stubborn one to overcome. Repeated attempts to meet this problem have been made, but none with a degree of success until the perseverance of Fred S. Meyer, of Minneapolis, at last gave us the first real improvement on Dr. Taggart's discovery of twenty years ago.

Dr. Meyer's work on this phase of inlay casting began shortly after the first paper and demonstration by Dr. Taggart, and his first findings were published in 1912, in the *Dental Cosmos*. C. S. Van Horn of Bloomsburg, Pa., as well as several others, have contributed to this subject. But it was not given to the profession on a sound working basis until Dr. Meyer completed the technic which made it possible.

Regardless of all other contributors, let it be understood that none have dared to come before a dental society and do what many of you saw him do here this afternoon, which was possible only after years of painstaking effort and personal sacrifice.

"Let us remember that mankind is helped in its progress almost as much by the study of imperfection as by the contemplation of perfection. Had we nothing before us in our futile and halting lives but saints and the ideal, we might well

fail altogether. We grope blindly along the catacombs of the world, we climb the dark ladder of life, we feel our way through futurity, but we can scarcely see an inch around or before us. We stumble and falter and fall, our hands and knees are bruised and sore, and we look up for light and guidance. Could we see nothing but distant, unapproachable impeccability, we might well sink prostrate in the hopelessness of emulation, and the weariness of despair. Is it not then, when all seems blank and lightless, when strength and courage flag, and when perfection seems remote as a star, is it not then that imperfection helps us? When we see that the greatest and choicest images of God have had their weaknesses like ours, their hour of darkness, their bloody sweat, are we not encouraged by their lapses and catastrophes to find energy for one more effort, one more struggle? Where they failed, we feel it less dishonor to fail. Their errors and sorrows make, as it were, an easier ascent from infinite imperfection to infinite perfection."

# GOLD INLAYS*

By ELMER J. SUNDBY, D.D.S., Duluth, Minn.

BEFORE I enter into a discussion of my subject matter, let us all bear in mind the necessity of correct cavity preparation in all our work. It is of the utmost importance. More failures are due to carelessly and improperly prepared cavities than to ill-fitting restorations made by a poor waxing and casting technic. This is especially true of the three-quarter type of abutment in bridgework.

We must seek as nearly parallel walls as will permit the withdrawal of the wax pattern with ease. Parallel walls greatly increase the frictional resistance to dislodgment, and a great saving of tooth structure is the result. A much shallower cavity can be prepared, with less chance for the casting to become loosened by the stress of mastication.

Waxes have always been a great source of trouble in our casting processes, owing to the fact that they expand on heating and contract on cooling. The degree of contraction and expansion is approximately 0.1 per cent for every 5 degrees change in temperature, this depending on the wax used. This change in the wax has worked to our disadvantage in the past, but in the wax expansion method, which Dr. Meyer has perfected, these former disadvantages become distinct advantages and are controlled by the elements of heat and cold to such an extent that the expansion of the wax, together with the ability of the investment to resist shrinkage, properly compensates for the shrinkage of gold in the process of casting.

It is more desirable to use the direct method of impression taking whenever possible; first, because a more accurate pattern can be obtained, and, secondly, because it saves time. But, after all, what we are seeking is a pattern as nearly perfect as possible. One man may obtain results one way; the man next door, another way. I am indebted to Dr. Meyer

*Read before the Chicago Dental Society, Nov. 19, 1929.

for the method which I am about to describe. It has been a great help to me.

### DIRECT METHOD FOR THE MAKING OF A MESIOCLUSODISTAL WAX PATTERN

A mesioclusodistal pattern has been selected because it is not easy to make a perfect one. Imperfect gingival margins usually show up on step fillings on bicuspids and molars of this type. You may soften the wax in your own way, with hot water or dry heat. If dry heat is used, care should be exercised not to overheat the wax until it runs. The wax loses its essential characteristics and be comes mushy. When the wax has been shaped to meet the requirements of the cavity, the portion that is to be placed against the gingival seat is slightly reheated and forced into the cavity immediately with considerable sustained pressure. The wax next to the fingers is slightly cooler and serves as a plunger. A generous piece of wax is used, as bulk is necessary and a goodly excess will do no harm. When this soft wax is forced into the tooth, there is a tendency on the part of the wax to flow away from the gingival margins. If the wax is carved flush with the tooth in this condition, the gingival margin is liable to be rounded. As soon as the wax is forced into the cavity, it is chilled with cold water, and the excess of wax that has overlapped the tooth is removed on the buccal and lingual aspects, but is not trimmed at the gingival. A hot instrument is used to cut through the wax on the occlusal surface from buccal to lingual aspects somewhere between the points that will tend to bind when the inlay is cast. This will relieve some of the tension produced in the wax when chilled. When the wax again returns to the temperature of the mouth, it will expand some, and the pattern will fit loosely against the mesial and distal axial walls. This helps somewhat to relieve the inlay when cast. At this point, the occlusal structure is carved roughly, because the wax is firmly seated, and there is little chance for any distortion of the pattern. We are now ready to correct the gingival margin. There is a shyness there, and it must be corrected by some method.

The wax is cut out at the contact points, an opening sufficient to easily pass a matrix being left. The matrix between the wax and the adjoining tooth is so placed that the bulk of wax lies between the matrix and the gingival margin of the cavity, the matrix extending slightly beyond this margin. A warm instrument of suitable shape is placed between the matrix and the adjoining tooth opposite the gingival margin, warming the matrix and, through it, the wax. With the fingers of one hand, the matrix and the wax are held in position and, with a slight pressure on the heated instrument, held in the other hand, the matrix is worked toward the tooth, the softened wax being forced into the cavity. This procedure will fill in and completely seal any defects at the gingival margin.

This method is preferable to the one in which the wax pattern is removed and softer wax added to the gingival portion before reseating. There is too great a chance of distorting the other margins of the pattern in removing and replacing. After the matrix is removed, there is a thin overlap of wax at the gingival margin. This is easily removed with any one of a number of wax carvers. An explorer is an excellent instrument for trimming the gingival margin if the excess of wax is not too thick. For the final finishing of this margin, a plain linen strip or dental floss can be used to good advantage.

The wax pattern is now ready to be carved in detail, and the other margins can be corrected, if necessary.

About three years ago, Dr. Meyer told me of a simple and sure way to correct a faulty wax margin. I can assure you, "it works." It is very unsatisfactory to try to melt wax into a crevice between a wax pattern and a tooth. Wax will not flow to any great distance and, where it does flow, it either pulls away from the tooth when it is moist or sticks to it when dry. A correction made in such a manner is often so superficial that we sometimes polish through it when finishing down the inlay.

A hot flat instrument is used to force it into the wax pattern about 1 or 2 mm. from the margin that is defective. When the wax has cooled slightly, but is still soft, it is forced toward the margin until it laps over it. Then the notch left by the hot instrument is filled in with melted wax and, when the wax is sufficiently cooled, the whole mass is compressed toward the tooth. The little overlap of wax is then easily finished down to the margin, and the wax will not stick to the tooth. As a final finish, a moist pledget of cotton is used to go over the occlusal surface and all accessible margins.

The heated sprue pin should be attached to the wax pattern while in the tooth, when possible, because the less the pattern is handled outside of the mouth, the better. Air is preferable to water for chilling the sprue pin and is easier on the patient and the pattern. After the pattern is withdrawn from the cavity, a sufficient amount of wax is added on the mesial and distal aspects for contacts. Some prefer to solder on for the contact after the casting is made. Either method is satisfactory. A sprue base should be in readiness, with a little beeswax in the hole and also in the groove on the sprue base. This wax is soft enough to permit forcing the sprue pin into it and hard enough to hold the sprue pin rigidly without any danger of its loosening, as is often the case when the wax pattern is being painted with investment. The beeswax in the groove on the sprue base will seal the joint when the investment ring is forced into it. This prevents any water or investment from leaking out and enables one to pick up the mold safely by grasping the ring. When the wax pattern is securely mounted in the sprue base, it is invested immediately to avoid distortion or contamination.

### INDIRECT METHOD OF IMPRESSION TAKING

I rarely use the indirect method for mesioclusodistal inlays or smaller castings. Occasionally, if the cavity is located in a rather inaccessible tooth or other unfavorable conditions present themselves, the indirect method is resorted to, but never purely indirect; indirect-direct, rather. The wax pattern is always checked on the tooth in the mouth after it has been waxed up on the model.

As the technic is practically the same for a full cast crown by the indirect method as it is for an inlay, the full crown technic will be described. After the tooth has been prepared, with walls as nearly parallel as possible, a copper band is selected and fitted to the tooth. It should be long enough that, when it is put in position, it will go just beneath the gingival margin preparation. It should be slightly larger in circumference than the tooth at the gum line. The tooth should be moist; soapsuds makes an ideal lubricant. The copper band is filled with soft modeling compound; the surface that is to go next to the tooth is passed over a Bunsen flame and then into hot water, and the band is forced

into place with a firm, even pressure until it is in its proper position. A finger over the open end of the band serves as a stopper to force the modeling compound well against the tooth. The compound is chilled while under pressure, and removed.

It often happens on removing the impression that certain defects are found at the gingival margin, usually due to air pockets. These are easily corrected in this way: With a small instrument, these defects are filled in with hot compound, being corrected as nearly perfectly as possible or left a little deficient. The tooth is moistened again and the impression replaced on the tooth, cold. Never heat the impression before replacing. With the impression in place, a blast of hot air is thrown on all sides of the band until the compound softens enough that, under pressure, a little compound will be forced out at the gingival. It is then chilled and the correction will be nicely made. The impression is now ready to flow or pack, as you choose.

Before the wax expansion technic was available, it was necessary, in indirect work, to make models for full crowns and large restorations of amalgam, which enabled us to get a model sufficiently hard and tough enough to swage the castings into place, owing to the shrinkage of gold. This procedure required considerable time to prepare the impression before packing the amalgam, and a much longer wait was necessary before the amalgam was set sufficiently to proceed with the waxing of the crown. I have not used amalgam for indirect models in my practice for several years. Model "kryptex" meets all the requirements of the expansion technic. It sets in a few minutes and, as swaging is unnecessary, is sufficiently hard and accurate to wax to. The modeling compound impression can be prepared for kryptex in one minute by simply cutting a narrow strip of pink wax, 24 gage, about half an inch wide and wrapping it around the open end of the copper band and sealing with a hot instrument. This gives us an extension of the model, which, for convenience of handling, is quite necessary. The impression is now ready for filling with kryptex.

On a cold slab, the kryptex is mixed quite thin, and applied with a small brush or instrument, in one corner of the impression, which makes it flow ahead into all the crevices. It is then tapped, before filling. This will eliminate all bubbles. A kryptex model can be separated in about five minutes. If a wax bite is necessary, it can be taken, and no time is lost, as the model will be hard about as quickly as we can get the bite. It is convenient to use a piece of 60-gage tinfoil covered with beeswax, or any suitable wax, in taking a bite impression. A more nearly perfect reproduction of the teeth will be obtained when the impression is flowed. This is especially true in a close-bite case. The kryptex model is separated from the compound after softening in warm water. Should any compound stick to the model, it can be removed easily with a pledget of cotton dipped in chloroform. We now have a model of the tooth to be crowned, and a wax bite.

Next, the base of the kryptex model is ground slightly cone-shaped, a vertical slot being made in the base. It is then covered with a thin coating of melted wax or petrolatum (vaselin) and placed in its proper position in the bite. This slot is made to assure accurate seating in the plaster model, which is now flowed up and mounted in a small crown and bridge articulator, with quick setting plaster. The actual time required to carry this work from the impression to

the kryptex model mounted in the articulator is between fifteen and twenty minutes, so we can readily see the saving of time. It is not really necessary to try the wax pattern of the crown in the mouth, as the contacts, occlusion and general contour of the crown can all be quite accurately established on the articulator. As a check, it is safer to try the pattern in the mouth, before casting, to make any correction. Two sittings will suffice to complete a full cast crown. To prevent kryptex models becoming dried out, they should be kept in water. A more nearly perfect adaptation of the wax pattern is made possible by placing the kryptex model in warm water before waxing up.

Much could be said about tooth anatomy, but only continual study and serious application of our findings, by way of carving, will produce results.

To focus all of our attentions on the finished margins and neglect tooth-form function in our restorations is stopping far short of the goal we seek. No casting should ever be placed that does not harmoniously carry out Nature's mechanical plan for each human masticating mechanism. It is the most highly developed mechanical apparatus in the entire body, exhibiting a scheme of design that challenges our best efforts to reproduce. The curvature of the various surfaces of the tooth, the rounding cusps, marginal ridges with spillways, grooves and sulci, the buccal, lingual and occlusal embrasures, must all be properly placed to insure functional, mechanical and physiologic harmony in all ranges of occlusion.

No single factor in the harmony that goes to make up the whole mechanism is more important than the manipulation of wax for the castings of the various units.

# GOLD INLAYS*

By ERNEST H. GOERING, D.D.S., Duluth, Minn.

IN the past, the great effort expended in attempting to duplicate the wax pattern was attended with variables occurring in the materials used. Besides the physical properties of the wax, and the shrinkage of the gold from the liquid to the solid state, we had the changes taking place in the investment itself. These changes revolve chiefly around the binder in these investments, which is plaster of Paris.

This brief résumé on investments is familiar to nearly all of you. It is given along with the expansion technic in the hope that there may be a renewed interest in the problem of the responsibility for our failure to get consistently good results; and what is meant by this is not only do we want consistently smooth castings with sharp margins, but these castings must fit standardized models, such as porcelain teeth or steel dies. It is only in this way that we can be sure that we are not compressing the dentin of the tooth and causing serious injury to the pulp.

Besides plaster of Paris, which acts as the binder of all investments, we have

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the fillers added to make up the balance of the investment. The chief among these is silex, although, in some investments graphite, coloring material, etc., have been added. Their advantages and disadvantages will be brought out briefly.

In starting with the carbonization technic, I shall outline briefly what happens to the mold in getting rid of the wax and water. The mold being placed on the heat, we have first the elimination of the bulk of the wax and some of the water. Next, the water is entirely eliminated. When this moisture is gone, the mold starts to smoke, and it is this carbonization of the wax that formerly prevented our getting a casting when the moisture was gone. During the interval of waiting for the smoke to disappear, if the mold were kept on the heat, we would get a breaking-down of the plaster binder in the investment, causing a roughness in the gold casting. These castings would always be smoothest on the inside owing to the heat reaching the outside of the mold first and breaking down the investment, because of the driving off of the water of hydration in the plaster.

The American Gypsum Company has been kind enough to furnish some interesting data on plaster of Paris. The temperatures herein mentioned are all Fahrenheit.

Briefly, for practical purposes in inlay work, wet set plaster shows no disintegration below the boiling point of water, or 212 F. Plaster, having been set, may be subjected, for short periods, to temperatures higher than the boiling point as long as the casts are wet. If kept there for long periods of time, plaster starts to shrink and crack at about 150 F. Under ordinary conditions when it is used as an investment and the time of exposure is short, the shrinkage will occur between 200 and 600 F., but only after the free water in the casting is driven out and the water of hydration in the plaster is removed. In ordinary dental plaster, the expansion is approximately 0.2 per cent.

O. H. Harder[1] shows that plaster of Paris varies in shrinkage according to the heat to which it is subjected. At 1300 F., a shrinkage of more than 4 per cent was noted. At lower temperatures, the shrinkage was much less; for example, at 500 F., it was 0.6 per cent, and at 800, 2.4 per cent.

In tests on investment at 1300 F., shrinkages of about 1 per cent were noted. The addition of fillers, such as silex and graphite, has the tendency to cut down on the shrinkage of the plaster after it has been decomposed by heat. The data illustrated that, in plaster of Paris, we get a small initial expansion, but as the heat is applied, we get a gradual shrinkage as the temperature mounts.

Additional data by other writers show that, in some investments, we get expansion when the plaster is brought up on the heat, and then shrinkage. We get a key to eliminate at much lower temperature to prevent as much as possible these changes that take place in the plaster binder in the investment.

In the carbonization technic, it is advisable to have considerable filler in the investment to cut down on the shrinkage of the plaster. The mold is greatly weakened by the addition of these fillers and offers little resistance to shrinkage of the gold from the liquid to the solid state. To have a strong mold, we must have a higher plaster content investment or, better yet, some new material that will act as a binder and still not be so erratic when subjected to heat. This high plaster content investment would not stand up well in the carbonization technic.

1. Harder, O. H.: Dent. Cosmos, 68:1129 (Dec.) 1926.

I believe that Dr. Meyer has solved this problem with the use of the steam boiler to remove the wax. We can now use a high plaster content investment. It will resist shrinkage better and, with the wax out of the way, we have now only to eliminate the moisture, and the heat formerly needed to carbonize the wax is not necessary. We have eliminated that period of waiting for smoke to disappear after the moisture is gone as we had in carbonizing the wax.

The Meyer investment, inner and outer, was designed for use with this boiler and the expansion technic. The outer, which is slow setting, is mixed first. For crowns and large mesioclusodistal inlays, water at 160 F., with the plaster bowl and mechanical mixer and measuring device warmed, is added to room-temperature investment. This is mixed slightly, and the resulting temperature will be about from 115 to 120 F. The mixing bowl is set on the right-hand side of the furnace, where it is kept warm until the inner investment is put on the wax pattern.

The inner slow-setting investment is mixed to a heavy consistency with room temperature water and spatulated for five seconds in a mechanical mixer. It should be of a heavy creamy consistency. It is slightly rasped against the inside of the pattern and the whole pattern is covered to a depth of about one-eighth inch and filled down well over the crucible former.

The former has a small groove filled with beeswax. A hot ring, which has been setting on the furnace, is now placed on the crucible former. This melts the beeswax and makes a water-tight seal. The warm outer investment is again spatulated and run around this room temperature inner investment and the invested pattern and ring is set in a water-bath at from 115 to 120 F., and allowed to set for forty minutes.

This is the correct temperature for the most difficult work. For smaller inlays, mesioclusodistal on bicuspids or three-quarter crowns on molars and bicuspids, less heat will be needed, 100 F. being ample. For small mesioclusal inlays, three-quarters on central and lateral incisors, very little is needed.

The steam boiler is a receptacle for water, containing a compartment on top with a screw cap to seat the inlay ring against a rubber washer. When the boiler is in operation, a metal apron catches the wax and water as it is removed from the mold. The boiler is filled with luke-warm water and the ring tightened into position with the screw cap. Small beads of water will be forced through the investment as the ring is securely sealed against the rubber washer. If these small beads of water do not come through, it needs more tightening, or we have trapped air in filling the boiler. These small beads show that everything is tight and ready to function when heat is applied.

The boiler is heated so that it will come to a boil in from five to six minutes. Small inlays, because of small surfaces, will boil out sooner and should be removed when the water in the top of the mold is just starting to boil. Large surfaced inlays and crowns should be boiled out until the sprue hole cannot be seen. The steam, condensing into water, washes out the wax. The boiler is chilled under tap water and the mold removed. It is now ready for elimination of the water.

We have the choice of two methods for completing this process. We can get a casting in from twelve to fourteen minutes by placing the mold with the sprue hole up in the compartment occupied by the boiler. The ring should be well centered here. This is high heat elimination.

The temperature is about 900 F. When a quickly disappearing cloud is seen in the cold mirror test, the mold for inlays should be removed and, when all moisture is gone, immediately cast. In making crowns, we can wait at least a minute after the moisture is gone and still get a smooth casting.

Bear in mind that this is high heat. Any carelessness here in carrying these molds too far will result in rough castings, owing to the breaking down of the investment. The second and safest procedure for all concerned is to eliminate the moisture at a temperature of about 300 F. for three and one-half hours, or longer. No mirror test is made here as it is not reliable. When three and one-half hours have elapsed, the casting can be made then or at our convenience. It can be left on this low heat all night without apparent damage. These molds can be invested and eliminated at our convenience, a week later if necessary. If we have used the boiler and eliminated the wax, we can safely let the mold stand a month in the laboratory. This will cause some drying-out and shorten the time of elimination a little.

With this low heat, we are, I believe, using the plaster of Paris to highest efficiency. We are getting a denser mold to resist shrinkage, and we are depending on this resistance, together with wax expansion, to produce as nearly an ideal result as possible.

Taggart's wax is used as a standard for this expansion technic.

There are a few additional points of interest which I should like to mention:

1. Never allow the mold to cool back to room temperature once it is ready. Always cast into a hot mold. Data on investments show further shrinkages when the mold is allowed to cool.

2. Tap water in most communities is chlorinated. This chlorin has a tendency to slow the setting of the investment. It is better to use distilled water on the first mix.

3. In using electricity, there is a variation in line voltage in all communities, depending on the line load. A thermometer should be used to assure that we are dealing with the temperatures as outlined.

4. Regardless of how well the finished casting fits, it should be relieved all over on the inside, except at the margins.

As the personal equation enters so greatly into everything we do, it is only with constant application that we are able to improve.

Having had the privilege of this association with Dr. Meyer in his expansion technic, I wish to thank him for giving me something that has improved my service to the innocents in the dental chair.

Further than that, we want to assure you that we will do everything in our power for further developments in our casting problems.

# GOLD INLAYS*

By F. S. MEYER, D.D.S., Minneapolis, Minn.

THE casting of a gold inlay or crown differs so little with all of us that it seems superfluous to say much of anything on the subject. The preparation for casting differs somewhat with the different technics, and advice on this subject may not be out of place.

In using any of the quick elimination or carbonization technics, there is a critical time at which the casting should be done in order to obtain the best results. It is therefore advisable to have the casting machine ready with the pressure adjusted if a pressure machine is used, and the spring wound up if a spring centrifugal machine is used. The gold should be cut up and ready and the blowpipe lit before the mold is placed in the machine.

If a quick elimination technic is used, a loss of time at this stage will allow the higher heat in the outside of the mold to penetrate to the inner surface, causing shrinkage or roughness of the casting, or both. In the slow elimination process, the temperature reaches a certain point and remains there. In such a case, the preparation for casting is just as necessary, for here a loss of time will allow the mold to cool, which will produce shrinkage of the casting.

The two principal types of casting machines are the centrifugal and the pressure.

Both have their advantages and their disadvantages. With the centrifugal machine, there is no danger of overheating the mold; while, on the other hand, the mold might cool a little in melting the gold. It is possible to use a larger sprue, which facilitates the flow of gold. On the other hand, the first pressure of the molten gold is not toward the sprue, but at right angles to it; which means that the gold will cool somewhat before it reaches the sprue, which creates a tendency to rounded margins.

This is especially true in the spring type of centrifugal machine. In my hands, that type of centrifugal machine has not been successful in casting crowns. With smaller castings, it has produced the most beautiful results. Others who use the centrifugal machine have been very successful and especially is that true with those using the type starting with a crank. Those of you who saw the castings made by Elmer Sundby of Duluth will agree with me that they can never be improved on with any pressure machine. On the other hand, you can say the same thing about the castings made by Drs. Vorhees of Chicago and Goering of Duluth. They can never be bettered by the centrifugal machine.

With the pressure machine, the initial force of the molten gold is directly into the sprue, but a smaller sprue is necessary to prevent the molten gold from dropping into the mold before the pressure is applied. Here, there is some danger of overheating the mold if too much time is utilized in melting the gold; and here,

*Read before the Chicago Dental Society, Nov. 19, 1929.

therefore, the question of a blowpipe must be given some consideration.

Any good blowpipe which will melt the gold in a reasonable time will do. If the flame is not hot enough, the mold may be overheated before the gold melts.

That is, of course, not true in case of a centrifugal machine when the gold is melted in a separate crucible. If the flame is too hot, care must be taken to avoid overheating the gold. At present, I am using an oxygen and gas blowpipe.

In my hands, with this blowpipe and a brush flame, I feel that there is less oxidation of the gold and less overheating of the mold than with a gas flame. Some caution is necessary where small quantities of gold are melted, and especially is this true when a pressure machine is used.

It is plain to see from the foregoing that the personal equation enters largely into the act of melting gold and casting. Some men melt their gold into a higher state of fluidity and use a lower pressure; some do the opposite.

I am using from 7 to 10 pounds pressure in my casting, and I cast the moment that the gold starts to roll around, or when the film which forms over the surface of the molten gold begins to roll back and forth.

The question of when to cast or the pressure to use must really be settled by the individual who does the casting. Experience is the guiding point, and one man's experience is of little value to another. I might say that with a centrifugal machine, a higher state of fluidity is necessary because of the longer time necessary for the gold to enter the mold.

The question is often asked, "What do you consider the best gold?" There are many good brands of gold, differing but slightly in color, hardness and strength. There are also some golds of such low grade that they are brittle and turn black in some mouths. Those, of course, it is wise for us to leave alone. As for the right gold to use for each particular case, that is a thing for each man to decide for himself.

The harder golds have some advantages and some disadvantages and so have the softer golds. As a general rule, we should choose the gold that is just hard enough to stand up under the particular stress that it is put to in each particular case. For instance, an occlusal filling does not require so hard a gold as a mesioclusodistal filling. A bulky mesioclusodistal inlay does not require so hard a gold as one with a thin occlusal filling which might spread at the gingival aspect on cementation. A mesioclusodistal where the cusps are shod does not require so hard a gold as one which is built up against a delicate cusp. In the latter case, the hammering of the gold by the force of mastication may cause a flow and hence a pressure against the weak cusp, and, in time, a fracture. The thinner the gold protecting a porcelain facing in bridgework, the harder must be the gold.

A prominent dentist confided to me two years ago that he was discouraged with porcelain root tips, that he had all together too many facings break. When I found that he was using a No. 1 gold for his dummies, I said, "That is the trouble." A No. 2 is much harder and it will stand the stress of mastication without flowing. I am not saying this to discourage the use of a No. 1 gold, for it is one of the finest for inlays and crowns, but it has its limitations. Too soft golds have been used for bridgework, for two reasons. In the first place, the color is better. In the second place, it could be burnished slightly to cover defects due to poor fitting castings. This is not only true of dental laboratories, but it also has saved the day for most of us at different times in

the past when our inlays did not fit. A dentist once said that a good dentist was not necessarily one that could set a perfect fitting inlay but one that could make a perfect fitting inlay out of one that did not fit. And that is what we have all been trying to do for the last twenty years. I am sorry to say that I have not always succeeded as well as I should have liked to. As for color, a gold has now been brought out that has the color of No. 1 and the hardness of No. 2.

The burnishing of our castings to make them fit should be a thing of the past, for all casting technics have been brought to a higher standard in the past two years.

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# MISPLACED UPPER LEFT THIRD MOLAR: REPORT OF CASE

By T. J. GEREN, D.D.S., Groesbeck, Texas

THE patient, a boy, aged 18, complained of feeling tired, suffered at times from insomnia, occasionally had a slight headache, was about 10 pounds underweight and had a sallow complexion, and a varying appetite. He had no pain.

The first indication of the source of trouble was the exodus of pus into the mouth coming from between the process and gums on the distal surface of the second molar. There was no soreness. Roentgen-ray examination of the teeth revealed that the lower left third and upper and lower right third molars were impacted. The upper left third molar could not be located with the dental film. Extra-oral roentgenograms showed the upper left third molar to be located in the osteomaxillary opening or the meatus from the nose to the sinus close against the orbital opening. The tooth was located by J. H. Wood of Houston, Texas.

The tooth was removed June 9, 1930, by the Caldwell-Luc operation after blocking of the second division of the fifth cranial nerve. The desired opening having been secured, the Cameron light was inserted, and about one third of the occlusive surface of the crown could readily be seen. A straight bone chisel was passed through the opening and forced between the bone and crown on the nasal side, and thus the tooth was dislodged from the bone. Then, with a long hemostat forceps, the tooth was removed. It was found that the buccal wall of the sinus had been absorbed by the pus.

Misplaced upper left third molar, located between nasal bone and orbital opening. (Roentgenogram reversed.)

Later the three impacted teeth were removed. The patient recovered rapidly.

# The Journal of the American Dental Association

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C. N. JOHNSON, Editor

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## *Editorials*

### THE RULES OF THE GAME

In every game of life—and incidentally life itself is the greatest game of all—there are certain rules. Therefore, the game of editing a professional journal has rules which of necessity must govern it, else it will not function properly. Manuscript comes in from various and sundry sources prepared in various and sundry ways, and the problem of presenting it to the profession in anything like an orderly fashion is not so easy as it may seem. The gentle reader, and particularly the ungentle one, as his journal comes monthly to his desk, has little conception of what is involved in its preparation. Some readers—bless their hearts—appear to have the idea that a magic wand is waved and, presto, the magazine is serenely sailing through the mail. This is strictly not true. Others have the notion that if they get their manuscript in the editorial office the last thing Saturday night, there is no earthly reason why it should not appear early Monday morning, provided Monday chances to be the first of the month. They forget that the preparation for the November number begins in July or August — this editorial for instance is

being written the middle of August — and that at the last minute certain necessities in the makeup of the magazine may compel a switching of material, and a given article may be forced out to go in a subsequent issue. With the best intentions and the best efforts on the part of the editorial staff, there is sure to be some disappointment in store for the contributor.

But, by and large, most contributors are becoming better informed and much more considerate, and, by the same token, they are preparing their manuscript in a more acceptable fashion. Which leads to a few suggestions that may serve to clarify matters between the contributor and the editor, and may help both. One of the rules is that manuscript—to introduce an Irish bull—should be typewritten; though legible handwriting may be permissible under certain emergencies. The type should be double-spaced, and the original instead of a carbon copy should be sent to the editor. Strange as it may appear, most of the "copy" that comes to the editorial office must be edited before publication, and it is almost impossible to edit single spaced type, and so far as carbon copies are concerned, most of them are so indistinct that neither the editor nor the compositor can do much with them. A little reasonable cooperation on the part of contributors will help wonderfully in bringing out material satisfactorily.

When proof is submitted to an author, he should have some consideration for the expense involved in making alterations. No editor will ever demur when a palpable error is corrected or when, as the case occasionally happens, a definite change in the author's views has taken place since the paper was originally prepared, and an alteration becomes necessary to bring it to date. This may easily occur when an appreciable time has elapsed between the preparation of the paper and its publication, and, under such circumstances, every consideration should be shown the author. But it is an altogether different matter when an author recklessly tears the proof to pieces, as is sometimes done, merely to gratify a personal whim, and without the least thought of the equity of the procedure.

Every journal adopts certain rules to govern its style and nomenclature, and the contributor must submit to those rules or

take his wares elsewhere. No matter what his individual preferences may be he has no right to dictate to the editor in this particular, though he has the inherent privilege of withdrawing his paper from publication provided it is an original communication and has not been read before any society. In case it has been read to a society, it at once becomes the property of the society, and goes automatically to the official organ of that body. The author has no more jurisdiction over the paper except that which may be granted by the courtesy of the society and the editor. The contributor should also remember that in this day of the linotype, when the most trivial change is made in a word, or even when a comma is inserted, the entire line must be reset, and there is always the chance that in doing this other errors will creep in that were not present in the original setting. All of which strengthens the argument against making any more alterations in the proof than are absolutely necessary.

Speaking of the time that sometimes elapses between the acceptance of a paper and its appearance in print, there is frequently what may seem on the surface a just cause for complaint on the part of the author. It is disheartening to a contributor to wait for publication the length of time that he is sometimes obliged to do; but there are various reasons for this, most of which are not in any way the fault of the editor. For instance, this is what frequently happens: An essayist reads a paper before a society and it is turned over to the officers. It may or it may not come promptly to the editor, but the essayist always naturally assumes that it does. After due lapse of time, the editor is likely to receive from the essayist an inquiry—sometimes very patient, at others not so patient—asking why his paper has not appeared. Probably the editor has never heard of the paper, and an investigation discloses the fact that it is calmly reposing in a pigeon hole in someone's desk. Such things have happened in the past and they are likely to happen again. Officials of societies and of sections of societies are human. So also is the editor and, despite his best endeavor, delays will occur. Papers that are incomplete often come

to him from societies, papers minus illustrations when illustrations are mentioned in the text. The natural inference would be that the editor should write the officers or the essayist at once and chase the missing illustrations. Granted that this is what he *should* do, let us submit the proposition that with all his multitude and pressing duties, coupled with the fact just admitted that editors are very human, is it not the most natural thing in the world for him to reach for the papers that are complete and sidetrack the incomplete ones out of their regular order? Even then he makes mistakes and overlooks things that he should attend to, just like other folks—maybe more so.

Yes, editing is a great game, and we have given only a few of the rules. The only thing a conscientious editor can do is to keep his publication on his mind as nearly twenty-four hours a day as possible, and play the game as best he may. Even then he cannot please everybody, however much he would like to.

## THE RELIEF FUND

At a meeting in Denver, it was made plainly manifest that the Relief Fund must be materially increased if it is to fulfil the function for which it was organized. It has now been in operation long enough to demonstrate not only its benefits and its feasibility, but also its actual necessity. Our Association cannot afford in this enlightened age to permit any of its faithful members to go to the poor house or to die of want. The exigencies of life in the natural expectancy of such things make it inevitable that certain of our members shall find themselves in their declining days without the means of support, a fact that has already become too apparent. Before the Relief Fund was established, these unfortunate men were permitted to drift with the current, and some of them doubtless hit a rock before their allotted time or floated in a disabled and helpless manner till the tide finally swallowed them up.

The amount of happiness that has been spread since the fund has been in operation can be realized only by those in charge of its

administration. Surely we think that every member of the Association must feel like holding his head a bit higher when he contemplates what his organization is doing for its needy and deserving co-workers. Up to this date, more than forty of our members have received assistance, and the need is constantly increasing as our membership enlarges and a greater percentage of our men are approaching old age.

To meet this inevitable need, we must build up our Relief Fund till it reaches such proportions that the interest will keep pace with the insistent demands; and this can very easily be done if every member will only do his bit—a bit so very small, so really infinitesimal, that it is almost uncanny to think that there is a single member who will ignore or neglect it.

In about a month from now, the Christmas seals will be sent out, and it is none too early to begin to set aside that dollar—that one hundred cents (think of it!)—to buy seals to make someone happy. Ten cents a day for a little longer than a week, one cent a day for a trifle over three months, a half a cent a day for much less than a single season—just to help to make someone happy!! Yes, to make more than someone happy; to make at least two people happy—the donor and the recipient—for be it known that the one to get the greatest happiness from a gift is the giver.

Put aside that dollar this minute and have it ready. The wrench of prying it loose will all be gone by the time the Christmas seals are delivered. Thanks a thousand times.

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## A NEW HOME FOR THE AMERICAN DENTAL ASSOCIATION

Backed by the proper authority from the Board of Trustees and the House of Delegates, the Ad interim Committee met in Chicago, Sept. 9, 1930, and consummated a deal for a home for the organization. This is a project that has been under consideration for some time, and its fruition must be a source of satisfaction to everyone who is interested in the welfare of the Association. It will

stabilize its activities and conserve its material interests as nothing else could, and it will lend solidarity to our entire organization. Each member of the Association may feel a pride in the fact that he is a copartner in this great enterprise, and he may experience a sense of proprietorship that he never could as long as we were dependent on renting quarters for our Central Office.

The home is located at 212 East Superior Street, Chicago, in a splendid environment, and a description of the property and the details of its purchase will be published later. Meanwhile the Association may be congratulated on having taken the greatest forward step in its history.

## INCENTIVE

Mr. Thomas A. Edison is quoted as saying that we do not know a millionth part of anything. If that is true—and few people would have the temerity to question a statement from Mr. Edison—then just stop and consider what that means for the human race. Think of the opportunity for everyone of us to add 999,999 more ideas to any single bit of information we now possess. The thought is thrilling and provocative. What an opportunity we have here presented to us for mental expansion and material achievement. According to Mr. Edison we have merely been trifling with the fringe of our problems. Let us get down deeper into the heart of things and find all these hidden facts so that we shall be more efficient than we have ever been and get more out of life. Think of the incentive to clearer thought and broader vision. Think of the untapped reservoirs of knowledge that lie just before us beckoning us on to a fuller, a better and a happier life. If Mr. Edison's statement is taken at par it means more for the future development of our race than any single precept ever made by our wisest men.

# COUNCIL ON DENTAL THERAPEUTICS

## DENTIFRICES

The Council has authorized publication of the following statement:

SAMUEL M. GORDON, *Secretary.*

### FOREWORD

THE Council on Dental Therapeutics, appreciating that long established customs cannot be changed at once, will, for the time being, adopt a very liberal policy in regard to the composition of dentifrices. Its labors for the present will be directed toward a control of irresponsible statements made in dentifrice advertising. But it will apply with the greatest leniency compatible with consistency a liberal policy regarding the composition of this class of products.

Hence, the Council issues the following "specifications" for dentifrices. The "specifications" have been made with a view of not imposing any hardship on either dentifrice manufacturers or dentists who have found particular classes of dentifrices useful in their practice. For the time being, the Council deems it pertinent to point out that extravagant claims in public advertising on dentifrices do not react to the public weal or to the benefit of the profession.

Dentifrices are defined as preparations, such as pastes, powders and liquids, which aid in cleaning débris from tooth surfaces. They must not contain substances which are poisonous or which are harmful to the mucous membranes of the mouth or other parts of the alimentary canal or to the exposed surfaces of the teeth.

*Sialagogues and Enzymes.*—It is immaterial whether or not dentifrices contain sialagogues or enzymes acting on either proteins or carbohydrates.

A very slight effect which these ingredients may exert is too transient to have any therapeutic value. Therefore, the presence of such enzymes should not be emphasized in the advertising of the products, since they do not exert any action either beneficial or harmful.

*Acidity or Alkalinity.*—A dentifrice may be either slightly acid or alkaline in reaction provided its degree of acidity or alkalinity is not great enough to injure either tooth surfaces or mucous membranes. The chemical reaction of the saliva may be changed for only a short space of time by the use of either acid or alkaline preparations.

The degree of alteration (of the chemical reaction of saliva) varies between wide limits, not only in different individuals, but also in the same individual at different times of the day. Therefore, the effect of such types of medication on the salivary glands, saliva, mucous membranes, teeth or detritus is very probably insignificant. Furthermore, the effect (so-called) of acids acting as mucin coagulants,

or of alkalis acting as mucin solvents, is questionable; this by reason of the fact that the concentration of these chemicals which is necessary to effect these reactions, is higher than can be borne comfortably by the oral or dental tissues. The mucous membrane, as well as the tooth surfaces, does not tolerate any significant variation in degree of acidity or alkalinity.

*Soap.*—Dentifrices may contain soap.

*Calcification.*—Various compounds of the alkaline earths may or may not be present, but no emphasis should be placed in the advertising copy as to their presence in the dentifrice nor any claim be made that they possess any value toward calcification of tooth or bone substance.

*Astringents, Disinfectants, Deodorants, Germicides, Antiseptics, Etc.*—The use of astringents, disinfectants, deodorants, germicides, antiseptics or other similar substances is permitted in spite of their doubtful value, but no claim should be based upon the presence of such substances in the dentifrice.

It has been shown repeatedly that a thorough rinsing of the mouth following a vigorous brushing with a simple nonmedicated toothpaste will reduce the oral flora to a considerable degree, but this reduction is only temporary. The addition of various medicaments does not effect notably this result.

*Abrasives.*—An abrasive may or may not be used as a component part of a dentifrice. If abrasives are used, they should have a hardness which is not greater than that of cementum, dentin or enamel. They should be in the form of impalpable powder.

# BUREAU OF DENTAL HEALTH EDUCATION

## FACTS DEMANDING SERIOUS PROFESSIONAL CONSIDERATION AS DISCLOSED BY DENTAL CONTACT WITH THE COMMITTEE ON THE COSTS OF MEDICAL CARE

By H. E. PHILLIPS, D.D.S., Chicago, Ill.

ABOUT one hundred years ago, the machine age annihilated the old hand craft and guild system of producing goods. That same machine age, with mass production in mind and efficiency methods in hand, is continuing to seek out all the few nooks and crannies where individual methods remain and is changing, transforming, or making obsolete these former methods of production or service. At the present time, the chain store is annihilating the individual shop keeper as surely as industry destroyed the old hand craft system over a century ago. This same social, industrial and economic flow is reaching out to engulf the medical and dental professions, both highly individualized types of service.

I only have to mention the growth of industrial medicine and medical and dental clinics, the development of sickness insurance in the United States and the tremendous changes in professional service that have taken place in Europe to call to your mind what I mean by social, industrial and economic flow.

To know that powerful forces of change not professionally directed and without full regard for the present social values in private practice are moving toward the profession and not to make plans for meeting them is to invite catastrophe. A more extended discussion of this subject is to be found in "What Direction, Please?" by P. Armstrong, M.D., of the Metropolitan Life Insurance Company, and in the "Medical Revolution" by Glenn Frank, in the *Journal of Surgery, Gynecology and Obstetrics*. The "Statement of Principles" in the address by Dr. Wilbur presented to the Committee on the Costs of Medical Care also discusses this problem.

I once passed by a valley in California where an uncontrolled flood had wiped out villages and towns. It was a scene of sorrow and destruction. At another time, I stood on the banks of Jackson Lake, Wyoming, and was told that this lake was made by the construction of a dam, which held the flood waters in control and only released them when needed for irrigation. A few days later, I drove by fine farms where this water was di-

*The term "medical care" is understood to include dental care.

*Read before the Section on Mouth Hygiene and Preventive Dentistry at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 22, 1930.

rected to do man's bidding and to bring forth harvest. Will it be possible for the medical and dental professions, with other groups in America, to direct the coming forces of change so as to save for society and the public the values inherent in our private methods of practice? There are two factors that will have a direct and powerful bearing on the way these questions will be answered:

1. Strong, compact, efficient, professional organization with visioned, intelligent leadership and informed membership.

2. A constant use of the illuminating power inherent in carefully collected, tabulated and coordinated facts to indicate the need for social change and the trends in that direction as they relate to medical care.

Can the mass of people afford medical care? Is modern medical science available to the mass of the people?

The Committee on the Costs of Medical Care was created May 17, 1927, to conduct a study of the problems involved in the foregoing questions, to collect all the facts available and to present to the American public and to those professionally engaged in the various branches of medical practice practical plans for securing adequate medical service to all the people at costs reasonably proportionate to their individual and collective means. A five-year program was outlined with this aim in view.

The committee has been in existence for a little over three years. It was in existence for a year and a half before a member of the American Dental Association was appointed to membership. There are now three members of the American Dental Association on the committee, namely, R. H. Rudolph, Trustee of the American Dental Association, Arthur D. Black, dean of Northwestern Dental School, and I. Alfred Owre, dean of the School of Dentistry of Columbia University, has been a member of the Committee since its inception.

The committee has published six studies and contemplates publishing between ten and fifteen others.

The committee meets twice a year in Washington, D. C., and a large proportion of the members attend. The executive committee of seven members meets monthly. A staff of nine outstanding individuals is conducting the research studies. The committee is made up of fifty persons and includes physicians, dentists, nurses, public health officials, business men, club women, sociologists, economists and statesmen. Dr. Ray Lyman Wilbur, Secretary of the Interior, is chairman.

The following seem to be the most significant facts disclosed by dental contact with the committee:

1. Social forces are tending to bring about a change in medical and dental practice.

2. The medical and dental professions have a common interest in directing with others these forces, so that the best interests of the public will be served. (The best interests of the public are also the best interests of the professions.)

3. Full cooperation of the profession with the committee will provide opportunity for participation in this direction as far as the committee's plans are effective.

4. The studies of the committee indicate that authentic factual material is vitally necessary to a clear understanding of the problems involved.

5. Factual material in regard to the incidence and effect of dental disease is scarce and unreliable.

6. Factual material in regard to dental clinics, industrial dentistry, advertising

dentists, European dental service, salaries of dentists working in clinics and industrial plants and other material will be needed in the near future.

7. Only about 25 per cent of the people receive dental service. The problem of providing dental service for the other 75 per cent at a cost they can reasonably afford is the major problem of the dental profession today.

Referring to my first point, relating to the pressure of social forces: In my judgment, there are at least four directions from which pressure is being brought to bear on the health problem of the nation.

1. The pressure from within the profession itself in its effort to meet the health needs of the people. This pressure is evidenced by the scientific professional spirit which engages in research work, hospital care, public health activities, health education lectures, demonstrations, charity work and the giving of leadership to social changes in the organization of medical service. Dr. Olin West, secretary of the American Medical Association, says: "The great outstanding question before the medical profession today is a delivery of adequate scientific medical service to all the people, rich and poor, at a cost which can be reasonably met by them in their respective stations in life."

2. The tremendous interest in health manifested by large lay organizations, such as women's clubs, parent-teacher and service clubs, the Red Cross and tuberculosis associations. Of even greater significance is the interest shown by the farmers' organizations and also by newspapers and magazines. These represent the awakening of the rank and file of the public to the importance of health care. This second group exert pressure that is often manifested in ill-advised political effort at improvement in medical service.

3. The great industrial organizations that not only want sober workers (I think these large industrial organizations are largely responsible for prohibition), but want them healthy as well. Sick or unhealthy workers are a risk to profits, and modern business is eliminating all unnecessary risk; therefore, many industrial organizations have experimented with medical service and, judging by statements made by their leading spokesmen, they are ready to make many more experiments. This group have little regard for old customs or traditions.

4. The fourth group are the economists, sociologists, social workers, philanthropists and statesmen who look on the present lack of application of scientific medical care to the whole people as an indication of a social and cultural lag. This group should supply the social vision that will help coordinate the points of view of other groups. With tact and intelligence, they may succeed.

All these groups are represented on the Committee on the Costs of Medical Care. They approach the subject from different angles. It is to be hoped that together they can study the facts disclosed by the different investigations being undertaken and together work out plans for the solution of what Dr. Wilbur calls "a task of great difficulty and magnitude."

For a detailed statement of this task, I refer you to the "Five Year Program of the Committee on the Costs of Medical Care." This booklet will give you a complete analysis of the problem and also includes a list of all the studies to be undertaken.

I call your attention to the fact that this is the first time, historically, that this particular type of social problem has been studied by the method of fact find-

ing and analysis. Should this method prove successful for this occasion and the professions and the public accept the plans for the future delivery of medical care, it may well be that society has learned not only how to attain physical health but other valuable lessons also. By applying the same methods of fact finding and analysis to the solution of other social ills, say of unemployment and the like, our country may attain social health as well.

In September, 1931, the major portion of the studies will be finished and the facts tabulated and coordinated. They will be read and digested by the members of the committee, and copies will be sent to prominent men and women all over the country for criticism and comment. Then, it is planned to have the whole committee meet for several weeks to discuss the entire problem and later make recommendations to the profession and to the American people as to what changes in the delivery of medical service will be beneficial and of value to the whole population. These recommendations, if they can be formulated so that they have the backing of the entire committee, will probably be accepted by the public and put into practice within the next two or three decades. It is, therefore, imperative and important that the dental profession collect all the facts and data bearing on their particular phase of medical service, so that all the available information will be at hand when the meeting takes place in September, 1931.

Can you conceive of a huge arc light being turned on dentists' professional activities, disclosing their annual incomes, fees, methods of bookkeeping, length of time in practice, hours at the chair, number of patients cared for, etc.? This information is being collected through the use of a questionnaire which has been sent during the past six months to about 9,000 dentists in twenty different states. To date, approximately 52 per cent of these questionnaires have been returned. They are being edited before tabulation. A minimum return of 75 per cent will be necessary before the Committee on the Costs of Medical Care will consider the returns of sufficient magnitude to tabulate. Thus, each state will have to reach 75 per cent in order to receive a tabulation of their returns. In regard to income, you can be assured that the returns indicate that the majority of dentists are not being overpaid.

The results of this questionnaire will be invaluable to the dentists who will represent the dental profession at the September conference.

As stated in point 5, there is one field in which there is a great scarcity of authentic statistical material. You must realize that one of the most important questions asked is, "What is the extent of dental defects among our population?" On the answer to this question hinges the importance of dental service, and yet there is very little factual material available on this subject. The material there is is clouded by the fact that all sorts of examination cards and records were used and all sorts of persons made the examinations. One report may have been based on a careful examination by a trained dental examiner, and another may have been the result of a casual look into an open mouth by an inexperienced physician, nurse or teacher.

One of the first publications of the Committee on the Costs of Medical Care concerned the incidence of disease in the United States. The proof sheets of this were sent to all the members of the committee for criticism and comment before publication. Your dental committee eagerly scanned the pages: surely, here

would be collected adequate, scientific, up-to-date data on the incidence of dental disease, the most prevalent and most common disease that the children of men are heir to. Your committee scanned the pages. The data were meager and misleading. Here are several items: A report from the Detroit Department of Health stated that of 2,093 children of preschool age, nearly one-third had dental defects, and examinations of school children showed that 33 per cent had defective teeth. A report of the physical examinations of men in the draft for the World War showed 3 per cent with defects of the skin and teeth.

I need not tell this audience how misleading the figures given are and how far they are from what is actually the truth as we know it.

Mr. Mills, the research worker who had compiled the data, was communicated with. "Why is the data on dental disease so meager?" he was asked. He replied, "I have collected from every source I know of and have included all of value. Do you know where I can get more?"

So we set out to get more information in order that a more comprehensive picture of the prevalence and the effect of mouth diseases could be published. We went to Dr. Wandel of the Bureau of Dental Health Education of the American Dental Association and asked him to get this information out of his files and pass it on to the Committee on the Costs of Medical Care. He informed us that he did not have it, that material of the kind requested was not available and to his knowledge did not exist. He would try to collect it. So he wrote to many of the prominent men in the United States who he thought might have material on hand. I quote his letter and some of the answers:

Dear Doctor Blank:

A report is soon to be issued by the Committee on the Cost of Medical Care, relative to the incidence of disease in the United States. Perusal of the original draft of this report indicates very unsatisfactory data from the standpoint of dental disease. Upon request to those in charge of the publication of this report, they have generously consented to hold it up for about a week to permit the introduction of more satisfactory dental material. This is an urgent appeal to you to come to the immediate aid of the dental representatives on this Committee to enable them to give dentistry the representation, in this report, which it deserves.

Following is the character of the information it is hoped you will be able to furnish, in whole or in part:

*First:* Statistics, indicating the prevalence of dental disease in the mouths of school children in your city.

*Second:* Statistics, indicating the influence exerted by dental disease upon school children in relation to absence from school.

*Third:* Information, indicating the influence exerted by dental disease in the general health picture of school children. (Has it been noted, in any given specific number of instances, that the eradication of dental disease has resulted in cutting down the previous incidence of such conditions as: tonsilitis, heart disturbance, kidney disturbance, nervousness, underweight, poor behavior, headaches, retardation, etc.? We especially desire such information, but it must be based as near on facts as possible.)

*Fourth:* Have you any information pertaining to the pre-school child along the lines mentioned above in the information requested on the school child?

Prompt mailing of this information in the enclosed, self-addressed, stamped envelope will be greatly appreciated by the Committee for whom this office is making this request.

Very hastily,
Bureau of Dental Health Education.
By Geo. H. Wandel, D.D.S.,
*Supervisor.*

This letter, as before stated, was sent to prominent dentists all over the country. Following are some of the replies:

From C. Carroll Smith, Peoria, Ill.: "I am sorry that I have no tabulated list

of items regarding our pupils such as you ask for."

From a letter in answer to one sent to Harvey J. Burkhart, Rochester Dental Dispensary: "We have no statistics with reference to absence from school. We have no statistics pertaining to the preschool child. While there are no definite statistics on hand we are told by the principals, etc., etc."

From H. R. Wilson, of Cleveland, Ohio: "I am this summer preparing for publication a study of preschool children and further I will prepare the results of a five year study of preschool children which I believe will be of some value. I will send them to you as early as possible."

Dr. Hollister, of Pennsylvania, gave a paragraph of statistics that covered part of the information asked for, and then said: "I trust these figures will be of some value to you." He also referred us to Dr. Detlefson, of Philadelphia, stating: "You should be able to obtain some statistics of value from him."

Then from the Forsyth Dental Infirmary, "On Question 2, I am unable to give you any definite information. On question 3, I have no definite facts. I am sorry not to give you any more specific information. I do not quite know where you can procure this, perhaps at Atlanta." (Signed by Percy R. Howe.)

Miss Gladys Eyrich, supervisor of the Division of Mouth Hygiene for Mississippi: "Regretting that I cannot furnish the statistics to prove what we feel to be good results obtained, etc."

From the State University of Iowa, by Thomas A. Gardner: "Relative to questions 2, 3, and 4, we have no statistics available."

From Dr. Detlefson, of Philadelphia: "I am enclosing data to cover your first question. The statistics on the remaining questions are not yet available in any form that would pass legitimate objections. I wish I could give you more."

In reply to a letter requesting information especially in regard to the incidence of dental disease as a primary cause or factor in such conditions as rheumatism, heart lesions, kidney lesions, gallbladder disturbances, eye lesions, circulatory disturbances, etc., Boyd Gardner of the Mayo Clinic replied, "It is impossible for us to give you the information to which you make reference."

As you see, authentic, up-to-date statistical material did not and does not exist. How can the dental profession pretend to furnish scientific data under these conditions? I will state, at this time, that for dentistry the most important lesson to he learned from contact with the Committee on the Cost of Medical Care is the need for scientific data and statistics on all dental questions.

You must not conclude that Dr. Wandel did not get some additional data for Mr. Mills. He did succeed to some extent and a great number of the present data on the extent of dental defects contained in Publication 2 of the Committee on the Costs of Medical Care are the result of his efforts. Nor do I want you to conclude that anyone has been negligent in not securing these data. Conditions had never made it necessary. Mr. Mills says that there are no complete statistics on health available. In the next few years, statistics will be increasingly necessary.

One of the most illuminating studies published to date by the committee is known as the "Abstract of Publication No. 6, A Survey of the Medical Facilities of Shelby County, Indiana," by Allon Peebles, Ph.D.

There are 25,000 people in Shelby County and 10,500 of them live in the

town of Shelbyville, the county seat. I quote: "The people of Shelby County spend over half a million dollars for the care and prevention of illness, about $21.00 per capita. Of this total, one-third was received by physicians, one-third was spent for drugs and medicines and one-third was paid to dentists, nurses, hospitals and other agencies."

There are thirty-one physicians and thirteen dentists in the county. Half of these physicians received a net income of less than $3,066 and half more than that amount. Only one physician had a net income of over $10,000. The accompanying tabulation gives the net professional income of thirteen dentists in the county.

| | No. dentists |
|---|---|
| $ 500–$1,499 | 5 |
| $1,500–$2,499 | 0 |
| $2,500–$3,499 | 5 |
| $3,500–$4,499 | 2 |
| $4,500–$5,499 | 1 |

The net professional income of the dentists is illuminating. More illuminating and more significant is the statement that a majority of the people visit the dentist not for examination, but because they are having trouble with the teeth; that 90 per cent of the industrial workers come only for relief of pain, and, according to the dentists, only about half of these patients who come for relief of pain return for fillings or other service. The others do not return because they cannot afford it. And further, "The dentists generally agree that parents wait until their children have toothache before dental care is sought. As yet, the parents do not recognize that the first set of teeth are important and that the first permanent molars come in when the child is 6 years old." These teeth are commonly beyond repair.

In a school examination, 820 children had defective teeth, and only 221 of these received dental care—about one-fourth.

This report by Mr. Peebles should open the ears of the socially deaf and bring sight to the socially blind in the dental profession. No profession that remains deaf and blind to the implications and demands contained in this publication is fit to carry the torch of progress. Up to the present, our profession has had the torch in its keeping, but the near future will demand, in larger measure than ever before, professional vision, courage and initiative. Every dentist will have to move his individual thinking apparatus outside his 8 by 10 office and change it so that it can function in terms of social need. In the next few years, the public service and oral hygiene committees should multiply their activities and should call more members to function in this field. We should extend and expand this work, both to meet the public need and to gain experience and outlook for our profession. I feel sure that if our profession meets its obligations in the future, it will be largely through those who have gained experience and outlook in the oral health field.

The Committee on the Costs of Medical Care is going through with its program, and our profession must not default in doing its part. The Trustees and the House of Delegates of the American Dental Association have shown vision and understanding of the work of the committee and have given full cooperation in all the dental studies so far inaugurated. They appropriated $7,500 last year for the survey now being made of dental practice.

If we are to meet our future obligations, we must gather much more information on other social phases of dentistry. This will require the further expenditure

of funds. If they are available, they will, I feel sure, be forthcoming.

The splendid response given by the dental profession in filling out the questionnaires and by the state officers and different committeemen all over the country in spending much valuable time in writing and telephoning in order to explain the need for the returns leads me to believe that the same spirit of cooperation will govern our response to any plan that the Committee on the Costs of Medical Care may present for giving adequate dental care to the people of the United States.

This committee collects and sends out to its members many articles published in both lay and professional magazines and bulletins, and also many pamphlets. For the dentist who is interested in this subject, the following articles are listed:

Cost of Living of Federal Employees in Five Cities, from the Government Printing Office. (See p. 246.)

What Direction, Please? by Donald B. Armstrong, M. D., Metropolitan Life Insurance Company.

The Status of Dentistry in Our Economic System, by Michael M. Davis. Reprint from THE JOURNAL.

Dental Care in the Family Budget, by Michael M. Davis. Reprint from THE JOURNAL.

The Medical Revolution, by Glenn Frank, Journal of Surgery, Gynecology and Obstetrics.

Economic Status and Health, Public Health Bulletin No. 165.

Medical Alliance Review, November, 1929.

Cost of Medical Care, by Lee Frankel, Metropolitan Life Insurance Company.

Articles in the Bulletin of the American College of Surgeons, December, 1929. (Pp. 9 to 28.)

Articles in the Bulletin of the American Hospital Association, January, 1930. (Pp. 60-72.)

I will append a list of the publications of the committee. Anyone interested may send to the Committee on the Costs of Medical Care, 910 Seventeenth St., N. W., Washington, D. C., for a copy of any one or all of these booklets.

Publication No. 1: The Five Year Program of the Committee on the Costs of Medical Care.

Publication No. 2: The Extent of Illness and of Physical and Mental Defects Prevailing in the United States, by Alden B. Mills.

Publication No. 3: Medical Facilities in the United States, by Allon Peebles.

Publication No. 4: Hospital Service for Patients of Moderate Means, by Niles Carpenter.

Publication No. 5: Medical Care for 15,000 Workers and Their Families (Endicott-Johnson workers), by Niles Carpenter.

Publication No. 6: A Survey of the Medical Facilities of Shelby County, Indiana, 1929, by Allon Peebles.

Special: Two Years' Work of the Committee on the Costs of Medical Care.

Special: The First Three Years' Work of the Committee on the Costs of Medical Care. An Address by Ray Lyman Wilbur, M. D., Chairman.

# PRACTICAL NUTRITIONAL SUGGESTIONS FOR DENTISTS*

By PERCY R. HOWE, D.M.D., Boston, Mass.

SINCE the teeth are modified bone, the matter of their sound formation and preservation is essentially one of calcium metabolism and fixation.

According to Sherman, modern diets are more deficient in calcium than any other food element. Therefore, the first object is to see that diets which contain ample amounts of calcium, such as milk, vegetables, fruits, are given.

The requirements of the growing child are several times greater than those of the adult per unit of weight. In consequence, infants and children especially need such foods. In order that infants may get an ample supply of human milk of a good quality, the prospective mother should be given calcium-containing foods.

It is understood that, according to natural laws, human milk is superior to any other food for the infant. Cases of rachitic conditions among babies reared on mother's milk have been reported, but it is safe to infer that the real cause is in the poor quality of the milk. Some idea of the relative value of human milk, as compared with even so good a food as cow's milk, may be seen from the experiments of Blanberg, as quoted by Orr. Blanberg found that infants absorbed 82 per cent of minerals from human milk as against from 60 to 70 per cent from whole cow's milk, 54 per cent from dilute cow's milk and 32.55 per cent from artificial foods.

There is ample scientific support for the statement that the mineral elements are well utilized from fruits and vegetables, especially orange juice. It is well known also that the ingestion of milk, fruits and vegetables by the expectant mother has a direct effect on lactation, enabling her to furnish an abundant supply of normal milk for the infant during the tooth forming period.

The question is often asked as to whether additions of inorganic salts to the diet are utilized. It is quite certain that the inorganic salts are utilized to some extent when the diet is very deficient in its inorganic content. According to Holt, Courtney and Fales, calcium lactate and carbonate were utilized in some test cases, but calcium phosphate was not. In animal experimentation, the inorganic salts are supplied as a salt mixture and constitute practically the only source of these salts.

Many studies have been made on this subject, and it is generally agreed, I believe, that minerals, when added to the diet, are utilized to some degree, but only if the diet is deficient in this respect. There is no question that mineral elements are utilized to a much greater extent from foods than from any other source.

*Read before the Section on Mouth Hygiene and Preventive Dentistry at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 24, 1930.

Parathyroid gland has also been given for the purpose of augmenting calcium utilization. It seems that parathyroid gland may raise the blood calcium somewhat, but at the expense of the bones and teeth, the body drawing on the stored calcium of these tissues rather than taking it from the food.

Besides the calcium itself, other factors need to be considered, as the acid ash in the diet. Meats and cereals have an acid ash. The resultant acids are sulphuric, hydrochloric and phosphoric, all strong mineral acids.

It has been estimated that, on the basis of 1,000 calories, meat yields acid to neutralize which 85.38 c.c. of normal alkali is required; in like manner, wheat, corn and rice require 37.08, 26.62 and 25.36 c.c., respectively, of normal alkali for acid neutralization; while on the other hand, milk gives 26.90 basic reaction (basic ash); potatoes, 98.75; cabbage, 138.26; spinach, 999.15, and orange, 118.77.[1] It is evident that milk, fruits and vegetables are the foods yielding a basic ash, while meats and cereals yield an acid ash.

In our experimental studies, Dr. Logan found that while cats and dogs could tolerate large amounts of the acids of meats and cereals without any calcium disturbance, more ammonia being formed for neutralizing purposes, with guinea pigs and monkeys a large calcium loss was found following administration of the acids. Hence, for sound tooth and bone formation the basic foods are needed.

Now we have to consider not only the calcium in the food *per se* and food elements which may divert it from its proper utilization, but also its assimilation, since 90 per cent of all digestion takes place in the small intestine, and since the calcium here enters the system, we need to take into account, among other things, the state of the intestinal wall, especially after the child begins to take solid food.

In animals that receive a diet of a pasty consistency, it is found that the intestinal walls are usually coated with mucus and slime. The villi themselves at times also contain mucus and the musculature is atonic. Proper absorption cannot go on under these circumstances. When roughage is added to the diet, a distinct improvement follows. This can be very definitely observed in experimental animals, such as the monkey, and undoubtedly the beneficial action of coarse foods on the teeth is due fully as much to the improvement in calcium absorption from the digestive tract as it is to the direct effect on the teeth induced by their more vigorous use and by the changing action of such foods.

The main source of roughage is vegetables and cereals, vegetables being preferable.

It has already been stated that cereals should be kept low in the diet on account of their acid ash, but this does not mean that cereals need to be excluded from the diet, but merely that the proportion of cereals to fruits and vegetables should be such that the vegetables in the diet exceed the cereal content. Cereals, which are seeds, contain many valuable factors. Probably only a portion is digested, and hence acts to furnish an additional amount of roughage.

Vitamins A, C and D have been studied in their relation to sound bone and tooth formation. Vitamin A, commonly called fat-soluble A, was at first thought to be a potent factor in calcium metabolism and in calcium fixation. As the vita-

1. Orr, J. B.: Brit. M. J., 2:504 (Sept.) 1924.

mins were studied more carefully, it was found that there was at least one other fat-soluble vitamin, now known as the D vitamin, which produced quite striking effects on tooth and bone formation.

The specific effect of the fat-soluble A vitamin seems to be exerted on epithelial cells, although, in general, other cell growth is retarded. The odontoblast reverts to the bone forming stage and erratic bone formation occurs in the pulp. Bone is laid down instead of dentin. Butter fat is an excellent source of the fat-soluble A vitamin. Other animal fats also contain it. Cod-liver oil is rich in this fat. In vegetables, it is found both in the leaf and in the storage organs, although here it is not associated with fat. It is also found in fruits. Grains also contain a certain amount.

The D vitamin, or antirachitic vitamin, has received especial attention. Cod-liver oil has been studied a great deal in this connection, and it has been found that the D vitamin is in some way related to ergosterol. This substance, in a purified form, possesses no calcifying property, but when it is subject to irradiation by ultraviolet light, it takes on very active calcifying properties, estimated by Bills to be 200,000 times greater than that of cod-liver oil. Such a powerful calcifying agent should be used with caution: too little is known of other effects that may accompany its specific action. Whether the process of calcification induced by the ingestion of irradiated ergosterol is confined to the bones, or may extend to other tissues, remains to be studied. Unquestionably, irradiated ergosterol has a place in the therapeutic field, but it should be employed with caution until its action is more fully understood.

In this connection, the matter of irradiated foods is to be considered. This is an artificial process for enhancing the antirachitic value of foods, and, according to experimental studies, notably those of Steinback, the antirachitic property of quite a wide variety of foods is greatly increased by the irradiation.

One would think that while this is an interesting academic fact, under the natural conditions of growth in sunlight foods would already possess this property. On the same basis, one might reason that the mineral content of food varied according to the soil, and it apparently does, and, hence, additional inorganic factors should be added.

I think it is safe, as a rule, to leave matters to nature. If fresh natural foods of the proper character are taken in sufficient quantity, the mineral elements and the vitamins will be abundantly supplied.

Vitamin D, essentially an antirachitic element, acts more during the formative period of the teeth than at any other time.

Rickets is in reality a cartilage condition. In rickets, the cartilage fails to degenerate normally; hence, the blood supply and osteoblasts do not enter the cartilage zone, and calcification does not proceed. Vitamin D causes the cartilage to degenerate and normal calcification to proceed. Much regarding the etiology of rickets remains to be more fully studied.

In practice, De Sanctis and Craig, from their study on infants, found that of 123 given viosterol, 29 per cent had rickets; while of 100 given cod-liver oil, 3 per cent had rickets. The conclusion of these writers is that other factors than vitamin D are responsible for rickets.[2]

A pint and a half of milk contains sufficient vitamin D for normal calcification, and I believe that it is better to furnish the system with this element by

2. De Sanctis, A. G., and Craig, J. D.: J.A.M.A., 94:1285 (April 26) 1930. Editorial, J.A.M.A., 95:38 (July 5) 1930.

means of the diet than therapeutically.

Vitamin C affects bones and teeth both during their formative period and afterwards. In a deficiency of this element, the odontoblasts cease to form dentin and the dentin already formed begins to liquefy. When the missing element is supplied, dentin formation is promptly resumed.

This element is found in all fresh, raw foods, and modern diets are extremely apt to be deficient in it. Fresh, raw milk contains it. Raw vegetables, and especially the leafy substances, are rich in it; while practically all fruits contain it. As modern diets go, I think that a pint of orange juice daily is needed to make good the deficiency.

It has sometimes been said that various vegetables contain substances which interfere with calcium utilization. Oxalic acid is one of these, for, in certain calcium tests, oxalic acid is used to precipitate calcium; but it is to be remembered that such a precipitation is a slow process and proceeds only under certain definite conditions. Such conditions are not at all likely to exist in the body. As far as I know, none of the ordinary vegetables and fruits act other than as excellent sources of mineral elements.

All laboratory findings support the idea that for sound tooth formation and subsequent tooth integrity, the wider use of milk, fruits and vegetables should be advocated.

## REPORT OF THE COMMITTEE ON STANDARDIZATION OF DENTAL EXAMINATIONS*

*The Committee on Standardization of Dental Examinations begs to submit the following report:*

ALL of the work of the committee has been done by correspondence. Only one meeting has been held for the purpose of assembling the report. The Committee believes that there is a definite need for standardization. Its first effort has been confined to the determination of principles and purposes of classification. No effort has been made to go into the intricate detail essential to a complete report on the subject. What we offer is only a working basis for future committee work in the form of a skeleton outline.

I. It is essential in the first place to classify the examiners in order to indicate the qualifications of the operators. They are listed as follows in the order of their efficiency: (1) dentist, (2) hygienist, (3) physician, (4) nurse, (5) teacher.

II. Dental examinations vary in completeness and detail in direct relation to the purpose for which they are conducted. A certain examination might be either inadequate or too cumbersome for a particular purpose. Moreover, the purpose of an examination closely determines the necessary instruments and technic, as well as the items to be recorded.

For these reasons, the Committee submits a classification of dental examinations based on the *purpose* of the examination, as follows: A. Research. B. Private practice. C. Hospital. D. School clinics. E. Industrial and other detached

*Read before the Section on Mouth Hygiene and Preventive Dentistry at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 22, 1930.

clinics. F. Survey. G. Other examinations, casual or incomplete, conducted largely for the purpose of education of the public.

III. Specified instruments and technic are as follows: Reference is made to a paper on classification of examinations for detection of dental caries by Dr. Alfred Walker of New York published in the *Dental Cosmos* for March, 1930.

Class A examinations: Dr. Walker's Type 1 requirements and in addition medical, social and other examinations, including a complete roentgen-ray examination and notation of clinical manifestations of oral pathologic processes, and laboratory tests demanded by medical and dental examinations.

B. The same requirements.

C. The same requirements.

D. Walker's Type 2 requirements modified in regard to tooth cleanliness. The teeth should be as clean as facilities will permit. Roentgen rays are employed where indicated.

E. Walker's Type 2 requirements with the addition of the use of the roentgen rays.

F. Purely oral examination; Walker's Type 3, noting in addition gross pathologic conditions and easily detected caries.

G. Instruments and technic not specified.

IV. Selection of data to be recorded has not been considered except casually and no suggestions or recommendations can at this time be made. The items will vary widely with the purpose of the examination and be limited by practical considerations.

V. All published examination data should specify as a preliminary a statement as to the class of examination employed and the qualifications of the examiner in conformity with whatever requirements may be adopted as standard.

VI. It is understood that this is only a partial report, submitted in tentative form and subject to change, and it may be considered merely as a report of progress.

Respectfully submitted,
S. W. MEEKER,
ALFRED WALKER,
LON W. MORREY,
FRANK A. DELABARRE,
*Chairman.*

## BRUSHING THE TEETH*

By C. N. JOHNSON, L.D.S., D.D.S., Chicago, Ill.

OUT of the more than 100,000,000 people in the United States, it would be difficult to estimate with accuracy the number who regularly use the toothbrush, but it would be much less difficult to make an estimate, among those who do use it, of the number who use it properly. The primary function of the toothbrush is not well understood by the rank and file of the people. There is one class, well represented by young ladies, who naturally wish to look beautiful and whose main incentive to brush the teeth is to make them look well. There

*Radio talk given at Washington, D. C., Oct. 10, 1929.

is another class, represented by the large majority of those who use the toothbrush, whose object, in addition to this, is to keep the teeth clean and prevent the formation of cavities in them. Each of these objects is quite commendable; but that is not all there is to the whole story of brushing the teeth. We use the toothbrush not only for the purpose of cleaning the teeth but also to keep the gums and supporting structures of the teeth healthy; in other words, to promote general mouth hygiene. If we lived on simple coarse food which compelled thorough mastication, we would not need to brush the teeth because the friction of the food would keep them clean, but most of our foods are so prepared that they do not call for vigorous mastication, and this, with the tendency to rush through meals which has grown to be the confirmed habit of the American people, leads to bolting the food, and thus the benefit that should come from the proper performance of the function of mastication is almost entirely lost. And the damage is not only to the teeth but also to the structures which surround them, because Nature intended that the teeth should be used for mastication and that the gums should receive vigorous massage by the food during this process. Lacking this, the gums usually become flabby and devoid of tone, and many cases of gum trouble or so-called pyorrhea may be attributed to this.

The fact that the real function of the toothbrush is to compensate for the lack of adequate mastication renders it important that we know how to use the brush properly, because its improper use may do much damage to the gums. When the attention of a person is concentrated solely on the idea of making the teeth white, and no consideration is given to the gums, there is too great a tendency to merely scrub back and forth across the teeth and neglect the gums entirely. The fact is that the gums need brushing as much as do the teeth, but they must be brushed in the right way. It is possible to injure the gums and force them away from the necks of the teeth by brushing too vigorously with a crosswise sweep of the brush, and this often leaves the necks of the teeth bare where Nature intended that they should be covered with the gum.

The gums must be brushed correctly. To give them the adequate amount of massage with the brush without injury, the upper gums and teeth should be brushed downward and the lower gums and teeth, upward. In other words, the force should be so applied that the brush is constantly pressing the gums against the necks of the teeth instead of away from them. When the gums are brushed in this way, the greater the force and the longer the brushing is kept up, the better it will be for the health of the mouth. To do this most effectively, the brush should be given a rolling motion against the gums and teeth with the bristles coming in contact with the upper teeth on the downward trip of the brush and against the lower teeth on the upward trip.

If you take a mirror and look in your mouth, you will see that between the teeth at the gum line there are little interstices in which food may lodge, to decompose and bring about decay of the enamel, and cause irritation to the gum. It is therefore necessary to dislodge all such material from these spaces, and the ordinary cross-brushing of the teeth does not accomplish this. It is in this capacity that the rolling or rotary motion of the brush is most useful, throwing the bristles into these spaces and freeing them from

any foreign material at the same time that it massages the gums most effectively. In this connection, the bristles should be driven between the teeth with a vibratory motion and with considerable force so that the surfaces of the enamel are polished clean and the gum margin is stimulated to healthy action.

With this kind of friction on the gums and teeth, we may, to a certain degree, make up for the loss of function through our insufficient mastication, and thereby maintain the mouth in a normal condition. But the proverbial "lick and a promise" will never do for the proper function of brushing the teeth. If we consider the aggregate loss of stimulation to the gums through failure to masticate, we shall see that the use of the brush a minute or two once or twice a day will never make up for this deficiency.

With nearly every individual, the process of brushing the teeth is altogether too incidental and perfunctory—it merely resolves itself into trying to whiten the teeth and letting it go at that. Brushing the gums to stimulate them to healthy function never seems to enter the mind, and as for taking a watch and timing the process till at least three minutes are spent on the teeth after each meal—well, this will at once be put down as a vagary of a man who has nothing on his mind but teeth. And yet anything short of this cannot properly be dignified by the phrase, "brushing the teeth."

Manifestly, in a limited radio talk, much must be overlooked pertaining to this subject, but I should like to refer briefly to one small matter. The question is frequently raised as to the virtue or otherwise of the various tooth pastes and powders on the market, and the intimation is made that they have no value; in other words, that the use of the brush and water is all sufficient. Doubtless, many of the claims made by the venders of these products are exaggerated—these venders are human just as you and I are—but I am not willing to admit that pastes and powders are without virtue. I firmly believe that the mouth is left cleaner and more wholesome by their use than without them, and personally I would almost as soon attempt to wash my hands and face without soap as I would to brush my teeth without a paste or a powder. It all resolves itself into a question of cleanliness, and these products help us to keep the teeth clean just as soap helps us to keep the hands and face clean. Use these products but do not expect them to work miracles. They were never intended to do that. If I have not made myself clear in this brief talk, a letter to the American Dental Association will bring immediate response. Please write to 58 East Washington St., Chicago, Ill.

# CURRENT LITERATURE

## EXTRACTS AND ABSTRACTS

*Problem of Dental Caries:* As a result of dietary and therapeutic experiments undertaken by the Ann Arbor group and reported by Bunting, Hadley, Jay and Hard *(Am. J. Dis. Child.,* September, 1930), active dental caries was reduced to a negligible quantity in 433 children. Of the methods employed, dietary measures appeared to be the most important. Evidence was obtained in support of the view that dental caries is a specific infective process, the activity of which is dependent on certain metabolic states. The diets prescribed for the control of caries were well balanced, well fortified, adequate rations in which sugar was reduced to the minimum. The authors review the various theories advanced regarding the cause and control of caries, discarding them all in favor of the theory of an infective process. The problem, they felt, might be attacked by measures to determine the possible presence of an inhibitory principle in the saliva that is antagonistic to the infective organisms, *Bacillus acidophilus;* by a study of the relationship of diet and nutrition to the growth and activity of this organism in the mouth, or by a study of the effectiveness of various prophylactic and therapeutic procedures for eliminating *B. acidophilus* from the mouth. Experimentation was conducted on the basis of the second and third theories. Metaphen and hexylresorcinol were found to be the most effective therapeutic agents, the latter being more satisfactory in general than the former, particularly in the case of teeth covered with mucin plaques. Caries was most successfully attacked by a combined use of dietary measures and daily mouth hygiene employing hexylresorcinol. The authors conclude: "It must be borne in mind, however, that the studies that have been reviewed are still in their early stages and that much work still remains to be done before any definite conclusions may be drawn. The fact that dental caries is frequently checked by the feeding of a normal diet does not prove that the disease is wholly due to malnutrition. There are many factors in the metabolic state, which may be equally important in the control of dental caries. These questions may be solved only by well qualified workers in the fields of bacteriology, chemistry, nutrition and other allied sciences to which this problem is related."

*Vitamin A Deficiency in Monkeys:* Observations on seventeen monkeys are reported by Evelyn B. Tilden and Edgar G. Miller, Jr. (*J. Nutrition,* September, 1930) from the laboratory of the late Hydeo Noguchi, whose interest in the problem made possible the execution of the work, according to the authors. Eleven monkeys were kept until death on a ration containing only from 6 to 12 units of vitamin A daily, and six were maintained on a similar ration, except that it contained from 250 to 700 units of the vitamin a day. The rations were exactly similar, except that, in the case of the deficient ration, the butterfat used had been aerated for eight hours at 120 C. and contained too little vitamin A to be detected by quantitative tests in rats. The first symptom of illness noted in the monkeys receiving the low vitamin A ration was loss of weight, which was followed sooner or later by loss of appetite and finally by colitis before death. In one animal, extensive edema was present shortly before death. There was no xerophthalmia, sinusitis or mastoiditis and tongue abscesses were not found in any of the animals. Histologic study of the tissues of the monkeys showed keratinization of epithelial tissue in one or more sites in nine of the eleven animals. In a few instances, the cornification was extreme, but in no instance were the conjunctivae involved in the keratinizing process. Six monkeys on the control ration, with one exception, remained well and gained weight. One animal developed a nonfatal dysentery, from which it appeared to have

recovered when killed. The intestines showed no gross changes at necropsy, but microscopic examination revealed parasites in the intestinal wall. These parasites were not detected in the intestines of the animals on the deficient ration which succumbed to colitis.

*Death Following Alveolar Abscess:* The *Dental Surgeon,* September 13, reports and comments on a case recorded in the *British Medical Journal* by Digby Griffiths. The patient, a man, aged 25, was in good health and had had no previous illness except indigestion. He had disregarded advice to have his teeth attended to. An alveolar abscess developed in connection with a left mandibular tooth, and although obviously ill and in pain, the patient refused to have the tooth extracted. Two days later, the abscess burst and the symptoms were relieved. The evening of the third day thereafter, he was apparently well except for a slight headache, when he suddenly exclaimed regarding the pain in his head and fell unconscious. Breathing was stertorous and the patient was cyanosed and sweating. The temperature rose and the pulse was irregular and rapid. The pupils did not react to light, and there was rigidity of the neck and limbs. Death ensued in five hours. Cerebral abscess, with acute septic meningitis, was diagnosed, the alveolor abscess being regarded as the source of the trouble. Comment on the case is as follows: "It is, of course, common knowledge that such sequelae as this may arise from dental causes; but cases of such gravity are fortunately so rare that the individual practitioner may come to regard them almost as fairy stories. It is well, therefore, that the occasional example should be reported, as a reminder that such things not only can but actually do happen."

*Pyorrhea:* A small group of cases are reported by J. S. Fairchild (*Dent. Mag. & Oral Top.,* September, 1930) as indicative that elimination of pyorrhea must be based on a correction of the nutrition of the patient. The first patient, an expectant mother, in the fifth month of her pregnancy, had an acute case of pyorrhea for which it had been suggested that Rabisheck's operation be performed. The dentist recognized the symptoms of scurvy and suggested an alkali-forming diet, the physician, the husband of the patient, concurring. All signs of the pyorrhea had disappeared in a month, and in six years the patient has had only one carious tooth and no pyorrhea. Her child has sound teeth. During a second prognancy, there were no signs of pus present, but the gums were slightly deeper in color and the teeth were loosened. In a second case, the mother of two children had early lost her teeth and all members of her family had bad teeth. The first child was born when the mother was badly nourished and had the typical narrow arch and crowded and carious teeth. During her second pregnancy, the patient ate a much improved diet, including milk, eggs, wholewheat bread and lettuce. The second child had a well shaped arch and almost perfect teeth. The author concludes: "I am convinced that pyorrhea is not a disease, but simply an abnormal condition, and cannot be contracted by a healthy person, either by contact or inoculation. The removal of the teeth, except in extreme cases, is quite unjustified, and does little or no good to the patient, as the condition is systemic and still persists, making it almost impossible to construct an effective artificial denture because absorption of the alveolus continues until the cause is removed."

*Medico-Dentally Speaking:* If I had a son who wished to practice dentistry, I should insist that he complete, as a minimum, the premedical—not the predental requirements. I should explain to him that the fundamental sciences of anatomy, physiology, chemistry and pharmacology are as applicable to the treatment of the teeth and jaw bones as to the remaining parts. I should advise him to complete the first two years of the medical course before beginning the study of dentistry. I should attempt to save him from the embarrassment of reading on the top of his final examination in anatomy, "Dental students answer the first eight questions, medical students answer ten." I should advise him to secure his A.B., B.M., or M.D., but should not insist on this, as degrees do not necessarily denote erudition or culture. I should warn him about the evaluation of his credits for advanced work or degrees in the future. I should emphasize that correct diagnosis is the supreme achievement in health service. I should remind him that surgical judgment is greater than technique. I should expect him to have greater knowledge of preventive dentistry than a member of any other professional group. I should

expect him to be able to discuss all phases of bone phenomena with the orthopedic surgeon and possibly lead the discussion. I should point out that he should approach his research problems from the standpoint of pure science. I should like him to feel, insofar as his personal preparation is concerned, that he is able to take his equal place with those earnest workers engaged in the advancement of science.—Hugh W. MacMillan in *Dent. Survey,* September, 1930.

## Foreign Literature

*Fast-Setting Amalgam. (Robert Kronfeld, Ztschr. f. Stomatol., Vol. 28, No. 6, 1930):* The author reports his experiences with a fast-setting amalgam that was brought out by Schenk in Vienna two years ago. The amalgam hardens in thirty minutes. For the most part, this type of amalgam is used for making inlay models. It makes it possible to have an inlay finished two or three hours after taking a compound impression of the cavity. Thus, it is a great help to everyone making inlays by the indirect method. The same service is rendered by this amalgam in the making of models of three-quarter crowns and jacket crowns. In cases of inlays in very deep approximal cavities, where it is hard to get a sufficient impression of the gingival margins, the author recommends making a low gingival base out of fast-setting amalgam and after it has been finished, taking the final inlay impression. If teeth or roots with deep caries are prepared for crown work, it is advisable to fill and restore them with this type of amalgam, which enables the operator to make the final preparation at the same sitting. If one has to fill two approximal cavities in neighboring teeth, it is usually necessary to make one approximal filling, then dismiss the patient and make the other filling the next day in order to get a good contact point. By using the fast-setting amalgam, it is possible to make both fillings in one sitting. The fast-setting amalgam is especially good for work on small children, as it gives the operator the opportunity to finish the fillings before the patient is dismissed and prevents destruction of contact points by mastication in the hours following the operation.

Rudolf Kronfeld, Chicago.

*Fatal Osteomyelitis of the Mandible Following the Extraction of Teeth. (W. Bauer, Vierteljahrsschr. f. Zahnheilk., No. 2, 1930):* The author shows histologic sections through the mandible of two patients that died after the extraction of teeth. In the first case, the patient, a man, aged 30, died three days after the extraction of a lower third molar from a mediastinitis. In this case, the pathologic changes in the mandible are found only in the immediate neighborhood of the alveolus. Here, the marrow spaces are filled with leukocytes and the bone shows beginning resorption: the symptoms of a purulent ostitis. In the second case, an osteomyelitis of the entire mandible developed in a woman, aged 51, after the removal of the lower front teeth, and the patient died a year later from septicemia. The inflammation followed the mandibular canal and caused a neuritis of the mandibular nerve. The bone is partially replaced by granulation tissue; in part, new formation of bone takes place, and there is a tendency to localization and demarcation of the process. The latter fact is in accordance with the clinical experiences which indicate a conservative treatment in cases of osteomyelitis of the mandible.

Rudolf Kronfeld, Chicago.

*Contribution to the Knowledge of Cementoma. (A. W. Rywkind, Vierteljahrsschr. f. Zahnheilk., No. 2, 1930):* A hard tumor was found in the posterior part of the mandible of a man, aged 21, and, on removal, this tumor looked like a conglomeration of teeth joined by an irregular bonelike hard substance. Histologically, it could be seen that the second molar, a bicuspid, and a part of the third molar were fused together. Their form was irregular, and they showed a common pulp chamber and a common dentin body. In addition to these teeth, there were areas of calcified masses, part of which were built like cementicles, and part consisting of merely irregular calcified masses of fibers. It is the author's opinion that, by some injury or infection in early youth, the germs of these teeth were damaged, which caused an irregular arrangement of the hard substances and a pathologic overgrowth of the cementum.

Rudolf Kronfeld, Chicago.

# MISCELLANY

## BOOK REVIEWS

*A Text-Book of Histology.* By Alexander A. Maximow, Late Professor of Anatomy, University of Chicago. Completed and edited by William Bloom, Assistant Professor of Anatomy, University of Chicago. 833 pages; 604 illustrations, some in colors; price, $9.00. Published by W. B. Saunders Company, Philadelphia and London.

In the midst of preparing this book, Professor Maximow died, and at the earnest solicitation of those who had been given some insight into the character of the work, it was taken up by Professor Bloom and carried to completion. The loss of Professor Maximow at this particular stage of his career was an almost irreparable one, but the enthusiasm and earnestness with which his successor approached the task of completion has been partial compensation, and the scientific world may be congratulated on the outcome. We do not recall having seen any illustrations on the subject that in the perfection of their detail surpass the ones in this volume. It had been the purpose of Maximow to base his findings as far as possible on human material, and he had gathered much that was new, not only in the text but also in the illustrations. It is fortunate, therefore, that, owing to the devotion of his colleagues, this material is now made available.

The chapter of greatest interest to the dentist is naturally that on "the oral cavity and associated structures." It is necessary only to refer to one feature of this chapter, a very commendable feature in a book written not by a dentist but by an anatomist. The nomenclature of the teeth is according to the most approved usage, and conforms to the various reports of the Committee on Nomenclature of the American Dental Association. The consideration thus shown to the point of view of the dentist on the part of the anatomist is exceedingly encouraging, and it will bear fruit in a greater interest in the entire volume by the dental profession.

The book as a whole must commend itself to everyone interested in the subject, and it is a credit to all of those who had a hand in its preparation.

*A Text-Book of Operative Dentistry.* By William Harper Owen McGehee, D.D.S., M.D., Head of the Department of Operative Dentistry, Professor of Operative Technology and Secretary of the New York University College of Dentistry. 932 pages; 1040 illustrations; price, $10.00. Published by P. Blakiston's Son & Co., Inc., Philadelphia.

It is refreshing to find a new volume on the subject of operative dentistry. This department of our work has in recent years suffered some decline in interest, owing in large part to the fact that other lines of practice have been magnified in the professional mind. It is unnecessary to go into the reasons for this on the present occasion, but the fact remains that operative dentistry has been in a certain sense neglected, and when this occurs, it is a fact that our patients are not receiving the dental service to which they are entitled.

The present volume very logically starts with a consideration of diagnosis, a greatly needed adjunct to operative dentistry, and the real basis of success in practice. Eighty-two pages are given to this important subject. Then, with equal logic, the author considers oral hygiene and preventive dentistry; which after all is the goal toward which modern practice should tend. The prevention of dental disorders is the keynote to the successful management of the mouths of the people we serve, and no emphasis can be considered too great to enlist the attention of the profession. These two topics take up nearly one fourth of the entire volume, which is an indication of the thoroughness with which they are treated.

The rest of the book consists of a consideration of the routine technical procedures of instrumentation in the various operations of filling teeth, pulp treatment, etc. These operations are outlined in some detail, and the student and practitioner should have little trouble in following them. The book is

a worthy addition to the literature of the profession and sustains very acceptably the reputation of the author and publisher.

*Dental Health.* By George A. Swendiman, D.D.S. Illustrated; 121 pages; price, $3.00. Published by The Stratford Company, Boston, Mass.

The object of this book is to impart practical knowledge on dental health to the laity. Under the topic "bad habits of childhood," the author considers thumbsucking as it relates to malocclusion and makes some brief suggestions for its cure. He considers the temporary teeth as well as the permanent set, and then takes up decay of the teeth, and other diseases. In one case, he shows the swollen feet and hands of a patient, the condition supposedly resulting from leaving an infected root in the jaw, but this case seems to lack substantiation. With no more comment than the author makes, it is hardly justifiable to leave such an impression in the minds of the laity.

In certain places, it would seem that the author was almost too technical for the average lay person, but the illustrations are good and will help to make the text clear. The book is well printed and bound, and the reader will find much in it of value.

*Cross-Sections of Rural Health Progress.* Report of the Commonwealth Fund Child Health Demonstration in Rutherford County, Tennessee, 1924-1928. By Harry S. Mustard, M.D., Director, Rutherford County Demonstration. 230 pages. The Commonwealth Fund Division of Publications, New York, 1930.

*A Chapter on Child Health.* Report of the Commonwealth Fund Child Health Demonstration in Clarke County and Athens, Georgia, 1924-1928. 169 pages. The Commonwealth Fund Division of Publications, New York, 1930.

These two volumes are precisely what their titles imply. Each is a careful check-up of the health conditions in the respective districts indicated, and they contain much significant information. The fact that our large foundations are concerning themselves more and more with matters pertaining to public health is one of the most promising signs of the time, as it is also one of the best expressions of real philanthropy. When the information thus being gained is carried down in a tangible way to a concrete service of the people, it must inevitably raise the standards of existence wherever it is put in operation.

*Handbuch der Zahnheilkunde (Textbook of Dentistry).* By Julius Scheff, Vienna; fourth edition, revised and edited by Hans Pichler, Vienna. Vol. IV: Prosthetic Dentistry. By Alfred Gysi, Zurich, and Ludwig Koehler, Cologne. 566 pages; 546 illustrations. Published by Urban and Schwarzenberg, Vienna and Berlin, 1929.

Gysi's work on the theory and practice of prosthetic dentistry is well known and much appreciated in this country. The discussion of the different problems in this field, however, is far from being settled, and, therefore, it will be especially interesting for the specialist as well as for the dental practitioner to have Gysi's ideas and methods accessible in one volume. The first part of the book dealing with the mathematical analysis and graphic reproduction of the movement of the mandible can be fully appreciated only by one who has spent a lifetime studying this subject. Gysi gives the scientific basis for the construction of his "trubyte" articulator and anatoform teeth. The movement of the teeth in a denture, the relations of upper and lower jaw and arch under various conditions (natural and edentulous) and the action of the artificial teeth in biting and chewing the food are plainly analyzed and described. The second part of the book, dealing with the practical questions, is written by Ludwig Koehler, co-worker and former assistant of Gysi. Koehler gives a thorough description of his method of making a proper diagnosis and of judging the changes in and the amount of atrophy of the jaws, and details the preparation necessary to obtain a satisfactory base for the construction of dentures. In the case of the edentulous, he suggests using charts on which all necessary data are marked, such as general conditions, time of loss of the teeth and condition of the jaw tissues in the various areas. The different types of impression materials and their chemical and physical properties are described and are correlated to the practical needs. This chapter is followed by the procedure of taking impressions and the various methods used in this operation. The way in which Gysi takes the bite and puts the models into the articulator is illustrated by very clear and instructive pictures. The author emphasizes the special importance of grinding to obtain the proper occlusion, as

the greatest adhesion of a plate cannot be obtained if there is a slight mistake or carelessness in the arrangement of the occlusal planes. In addition to Gysi's methods and ideas, Koehler gives a comprehensive review of many other methods used in making impressions, taking the bite and setting up the artificial teeth. The advantages and disadvantages of these different methods, including the question of vulcanite vs. metal plate, are discussed thoroughly. The laboratory procedures, for example, vulcanizing, are illustrated from the most modern point of view, taking into consideration all possible variations and difficulties in the work. The last part of the volume deals with partial dentures, their construction and retention.

In summarizing, we must say that this work gives in a very comprehensive manner complete information concerning all practical and theoretical questions of prosthetics. The illustrative material is of such high quality that a mere study of it is instructive and helpful.

RUDOLF KRONFELD, Chicago.

## CORRESPONDENCE

### PYORRHEA ALVEOLARIS

*To the Editor:*

Much has been written and will, I suppose, continue to be written regarding pyorrhea, but I do feel that the apparent antagonism of present-day views is due to the fact that this subject is so frequently discussed without an agreed definition as to what is meant by the term "pyorrhea alveolaris." Too often, owing mainly to the modern craze for specializing, we clutch and treat symptoms as cause, whereas they are merely secondary, thus leaving the cause untouched and the vision blurred.

A study of the rise and fall of various nations is a wonderful tutor to those who would ponder over their message. Therefore, let us direct our attention to a brief consideration as to how the old civilizations fared when prosperity, with its attendant luxury, became part of their daily routine. We know that pyorrhea became rampant when the Egyptians, Greeks and Romans deserted the simple food of their forefathers. We also know that this scourge did not afflict the peasant classes, who continued to follow a simple food régime. Surely, therefore, facts *can* teach us something.

For many years, I have been firmly convinced that pyorrhea (that condition where the teeth are loose, with pus usually exuding from inflamed gums, foul breath, and often gastric symptoms, constipation, rheumatism and general malaise) was merely a local symptom of a general toxemia, usually resulting from years of continued dietetic errors. Belief is one thing, but proof belongs to a different category. Those sufferers prepared to cooperate in a determined manner to save the teeth and to return to a simple and more natural diet, combined with appropriate physical exercise, as well as certain dental treatment, usually reap the rewards of a complete cure. A more striking point is that, when pyorrhea disappears, so do many of the other general complaints from which the victims suffered. Those who are interested in this mode of treatment I would refer to the report of a case of mine which appeared in the *British Medical Journal,* December 24, 1927, and to the chapter on pyorrhea alveolaris in my recently published book, "Those Teeth of Yours," where the subject is discussed in detail.

My plea is: Do not extract all pyorrheic teeth on sight—wait until other methods have been explored and have failed. I do not refute the statement that there are cases in which all the teeth ought to be extracted, but I have yet to be persuaded that these should be sacrificed in the present prodigious numbers.

I am convinced that the state of the gums is one of the most valuable clinical symptoms available for indicating the success or failure of Nature's fight against disease, and that the cure of human suffering will not be achieved by a complicated process, but by a method so simple in its application as to be frequently ignored. In fact, we often miss the obvious—we cannot see the forest for the trees.

J. MENZIES CAMPBELL,
D.D.S., L.D.S., F.R.S.E.
14, Buckingham Terrace,
Glasgow, W2.

Sept. 3, 1930.

### A WORTHY CHARITY

*To the Editor:*

May I invite, through you, the attention of the readers of THE JOURNAL to a charity which merits their attention?

At Lucknow, India, there is being conducted by Hakim Zaki Sahib a free dental and medical dispensary. The organization, I have determined from East Indian students at the University of California, is a worthy one but it is practically without funds and needs badly equipment and books. You will be doing a great favor if you will forward instruments and books directly to the following address:

Hakim Zaki Sahib
Lucknow, India
Poor's Infirmary
Oudh, V. P.

Some of the books specially mentioned as needed are as follows:

Operative surgery
Legal medicine and toxicology
Tropical medicine
Practical psychology used in medicine
Dictionaries of the English language and particularly medical dictionaries
Botany and zoology
Physiology
Texts dealing with Johann Gregor Mendel and his theories of dominant and recessive characteristics as demonstrated in plant hybrids
Serology
Hygiene
Applied chemistry in medicine
Encyclopedia of medicine
Essentials of homeopathy
Operative dentistry
Prosthetic dentistry
Bacteriology
Dental pathology.

Hakim Zaki Sahib especially requires:

A sphygmodynamometer
Brass syringes
Trays
Hospital equipment.

May I point out that this material should be sent by parcel post, insured; otherwise, delivery is not certain.

JOHN A. MARSHALL,
University of California,
First and Parnassus Avenues,
San Francisco.

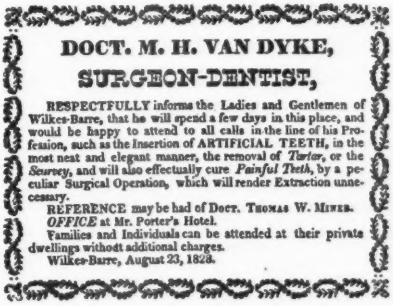
Sept. 12, 1930.

## NEWS

*Mr. Eastman Offers Sweden Dental Clinic:* The City of Stockholm, Sweden, has been offered $1,000,000 by Mr. George Eastman for the construction and equipping of a dental clinic similar to the Rochester Dental Dispensary. The Stockholm clinic will be the fourth established through gifts of Mr. Eastman, the others being the Rochester Dental Dispensary, which has been in operation since Oct. 15, 1917, the London Clinic, which is near completion, and the Rome Clinic, for which the ground has been broken. The primary purpose of the Stockholm institution will be to care for the teeth of poor children under 16 years of age residing in Stockholm and its suburbs. A clause in the contract which did not appear in the contracts drawn up for the London and Rome clinics provides that the Stockholm clinic shall establish and conduct a school for dental hygienists. Harvey J. Burkhart, director of the Rochester Dental Dispensary, who is now in London to attend the opening of the London clinic, will probably visit Stockholm to confer with the authorities there regarding the proposed clinic, before his return.

*Iowa State Society Elects Officers:* Following are the newly elected officers of the Iowa State Dental Society: president, H. J. Altrillisch, Dubuque; president elect, L. W. Snuggins, Des Moines; vice president, H. D. Coy, Hamburg; secretary, John Scholten, Cedar Rapids; treasurer, Frank Fourt, Fairfield.

*A Card of Historical Interest:* The original of the announcement card shown herewith is the property of C. J. Hollister, of Harrisburg, Pa., and was given to him by H. M. Beck of Wilkes-Barre, Pa.


DOCT. M. H. VAN DYKE,
SURGEON-DENTIST,

RESPECTFULLY informs the Ladies and Gentlemen of Wilkes-Barre, that he will spend a few days in this place, and would be happy to attend to all calls in the line of his Profession, such as the Insertion of ARTIFICIAL TEETH, in the most neat and elegant manner, the removal of *Tartar*, or the *Scurvey*, and will also effectually cure *Painful Teeth*, by a peculiar Surgical Operation, which will render Extraction unnecessary.

REFERENCE may be had of Doct. THOMAS W. MINER.

*OFFICE* at Mr. Porter's Hotel.

Families and Individuals can be attended at their private dwellings without additional charges.

Wilkes-Barre, August 23, 1823.

*Downward Trend of Infant Mortality:* Stock market crashes and unemployment have failed to disturb the downward trend of infant mortality in the cities of the United States during 1929, according to a report just issued by the American Child Health Association, in which the figure of 66.2 deaths among each thousand births is announced. Next to the rate of 64.9 attained in 1927, the rate for 1929 is the lowest ever recorded for the cities of the country. The decline has been almost continuous since 1915, when the Birth Registration Area, formed for the collection of dependable information, was organized. In 1928, the rate rose slightly to 68.3 over the low point of 64.9 in 1927. Fifteen years ago, the rate was near 100. Today, the rate is but two-thirds what it was just after the World War started. The 1929 rate was the lowest ever attained in Chicago, Philadelphia, Detroit, Boston and Baltimore. For the five year period 1916-1920, Pittsburgh exhibits the most outstanding decline, the rate dropping from 120 to 77 in the period 1925-1929. Portland, Ore., had the lowest rate among the cities over 250,000 population. Seattle was a close second with 46 and Minneapolis stood third with 49. Among the ten largest cities in the country, St. Louis and New York tied for first place with rate of 59. Close on their heels came the metropolis of the middle west, Chicago, with a rate of 60. Other figures were Cleveland 61, Philadelphia 62, Los Angeles 65, Detroit and Boston 69, Baltimore and Pittsburgh 73. St. Paul, Minn., was in the lead with a rate of 46 among the cities of the 100,000 to 250,000 class. Union City, N. J., stood first in the 50,000-100,000 class, with a rate of 25. In the 25,000-50,000 class, West New York, N. J., tied with Revere, Mass., each having rates of 26. Among the smallest cities with populations from 10,000 to 25,000, Northbridge, Mass., had the low rate of 15. As a group, the cities of the Pacific Coast continue their undisputed leadership as the banner home for babies. Oregon and Washington lead the procession with the cities of Minnesota in third place. Vermont, Utah and California cities show the same average rates.

## Deaths

*Basom, Joseph Newland,* Athens, Ohio; Ohio State University, College of Dentistry, 1925; died, June 8; aged 40. Dr. Basom was the son of Dr. J. S. Basom, with whom he was associated in practice. His widow, two daughters and an infant son survive.

*Eastman, William White,* Sonora, Calif.; died, July 11, after a stroke of paralysis; aged 69.

*Jackson, Robert C.,* San Francisco, Calif.; College of Physicians and Surgeons of San Francisco, a School of Dentistry, 1919; died, August 6.

*Johnson, Wallace,* Bluefield, W. Va.; University of Louisville, School of Dentistry 1907; died, September 11; aged 55.

*Nash, Benjamin C.,* New Rochelle, N. Y.; New York College of Dentistry, 1882; died, May 7; aged 72.

*Ottofield, Louis J.,* Maryville, Calif.; College of Physicians and Surgeons, of San Francisco, a School of Dentistry, 1924; died, July 24, at the home of his sister in Terre Haute, Ind.

*Perry, George B.,* Philadelphia, Pa.; Harvard University Dental School, 1891; died October 8; aged 69. Dr. Perry practiced in Chicago for many years.

*Welch, Dudley R.,* Chicago, Ill.; Chicago College of Dental Surgery, 1899; died, May 24; aged 54.

*Weller, Harry D.,* Indianapolis, Ind.; Indiana University School of Dentistry, 1896; died, July 23, after an operation; aged 55.

# ANNOUNCEMENTS*

## CALENDAR MEETINGS

American Dental Association, Memphis, Tenn., Oct. 19-23, 1931.

American Dental Society of Europe, Paris, France, July 29-Aug. 1, 1931.

Dental Protective Association of the United States, Chicago, Ill., Dec. 15, 1930.

International Association of Dental Research, Memphis, Tenn., March 23-26, 1931.

International Dental Congress, Paris, France, Aug. 3-8, 1931.

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Chicago Dental Society Midwinter Clinic, February 2-5.

Dallas Midwinter Clinic, February 16-18.

Kings County Midyear Meeting, Brooklyn, N. Y., February 25-28.

Greater New York December Meeting for Better Dentistry, December 1-5.

Southern Society of Orthodontists, Miami Beach, Fla., January 13-15.

## STATE SOCIETIES

District of Columbia, at George Washington University, Washington, D. C., second and fourth Tuesdays in each month from October to June, inclusive.

April (1931)

Kentucky, at Lexington (6-8).

Louisiana, at New Orleans, (20-22).

New Jersey, at Atlantic City (15-17).

May (1931)

Iowa, at Des Moines (5-7).

New York, at New York City (12-15).

Virginia, at Old Point Comfort (11-13).

June (1931)

Wisconsin, at Milwaukee (9-11).

## MEETINGS OF STATE BOARDS OF DENTAL EXAMINERS

California, at San Francisco, December 13, Bert Boyd, Secretary, 610 South Broadway, Los Angeles.

Connecticut, at Hartford, November 17-20. Almond J. Cutting, Recorder, Southington.

Delaware, at Wilmington, January 21-22. W. S. P. Combs, Secretary, Middletown.

Illinois, at Chicago, November 18-21. Horace J. Tharp, Secretary, 11112 S. Michigan Ave., Chicago.

Minnesota, at Minneapolis, December 12. W. H. Smith, Secretary, 2337 Central Ave., Minneapolis.

New Jersey, at Trenton, December 1-6. John C. Forsyth, Secretary, 146 W. State St., Trenton.

North Dakota, at Fargo, January 13-16. Gilbert Moskau, Secretary, Grand Forks.

Pennsylvania, at Philadelphia and Pittsburgh, December 10-13. W. A. McCready, Secretary, 1115 Highland Bldg., Pittsburgh.

Rhode Island, at Providence, December 2-4. Albert L. Midgley, Secretary, 1108 Union Trust Bldg., Providence.

Texas, at Dallas, December 3. Maxwell C. Murphy, Secretary, Temple.

Wisconsin, at Milwaukee, December 15-19. S. F. Donovan, Secretary, Tomah.

## DELAWARE BOARD OF DENTAL EXAMINERS

The Delaware Board of Dental Examiners will hold an examination for both dentists and oral hygienists in the Municipal Building, Tenth and King streets, Wilmington, January 21-22, from 9 a. m. to 5 p. m. All applications must be filed in the office of the secretary at least ten days before the date set for the examination. Full information, application blanks, etc., can be secured from

W. S. P. COMBS, *Secretary,*
Middletown.

## DENTAL PROTECTIVE ASSOCIATION OF THE UNITED STATES

The annual meeting of the Dental Protective Association of the United States will be held at the Palmer House, State and Monroe streets, Chicago, on the third Monday

*Announcements must be received by the fifth day of the month in order to be published in the forthcoming issue of THE JOURNAL.

in December (15), at 4 p. m. sharp. The reports of the officers will be given; a board of directors will be elected and such other business transacted as should come before the association. All members are urgently requested to be present.

By order of the Board of Directors,

J. G. REID, *President.*
D. M. GALLIE, *Vice Pres.-Treas.*
E. W. ELLIOTT, *Secretary.*

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## VIRGINIA STATE DENTAL ASSOCIATION

The next meeting of the Virginia State Dental Association will be held at Old Point Comfort, with headquarters at the Chamberlin-Vanderbilt Hotel, May 11-13.

A. M. WASH, *Sec'y-Treas.*
504 Medical Arts Bldg.,
Richmond.

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## LOUISIANA STATE DENTAL SOCIETY

The next annual meeting of the Louisiana State Dental Society will be held April 20-22, in New Orleans.

JULIAN S. BERNHARD, *Secretary,*
417 Medical Arts Bldg.,
Shreveport.

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## ALPHA OMEGA FRATERNITY

The annual convention of the Alpha Omega Fraternity will be held at the Hotel Statler, Detroit, Mich., December 22-24. Make reservations direct to hotel.

By order

WILLIAM RICH, *Supreme Chancellor,*
A. M. FLASCHNER, *Supreme Scribe,*
419 Boylston St.,
Boston, Mass.

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## DALLAS MIDWINTER DENTAL CLINIC

The Dallas Midwinter Dental Clinic will be held February 16-18. The clinicians are H. G. Morton, of Milwaukee (crowns and bridgework), and Arthur C. Engle, of St. Louis (surgery). A clinic on prosthetics will also be presented. There will be an exhibit by national manufacturers.

ROLAND LYNN,
1807 Medical Arts Bldg.,
Dallas.

## AMERICAN ASSOCIATION OF DENTAL SCHOOLS

The American Association of Dental Schools will hold its convention, March 23-26, at the Peabody Hotel, Memphis, Tenn.

R. S. VINSANT, *Sec'y-Treas.*
718 Union Ave.,
Memphis, Tenn.

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## INTERNATIONAL ASSOCIATION OF DENTAL RESEARCH

The International Association of Dental Research will meet in Memphis, Tenn., at the Peabody Hotel, March 21-22.

R. S. VINSANT, *Sec'y-Treas.*
718 Union Ave.,
Memphis, Tenn.

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## SOUTHERN SOCIETY OF ORTHODONTISTS

The eleventh annual meeting of the Southern Society of Orthodontists will be held at the New Fleetwood Hotel, Miami Beach, Fla., January 13-15. Among the essayists will be Alfred P. Rogers, Boston, Mass., A. LeRoy Johnson, New York City, Herbert A. Pullen, Buffalo, N. Y.; Frank M. Casto, Cleveland, Ohio, and W. W. McKibben, Miami, Fla. An invitation is extended to all members of the dental and medical professions to attend this meeting.

CARLTON B. MOTT, *President,*
Flatiron Bldg.,
Asheville, N. C.

OREN A. OLIVER, *Sec'y-Treas.*
Medical Arts Bldg.,
Nashville, Tenn.

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## FLORIDA DENTAL HYGIENISTS' ASSOCIATION

The Florida Dental Hygienists Association will hold its fourth annual meeting, November 14, at the McAllister Hotel, Miami. Members of the dental profession and all registered dental hygienists are urged to attend.

ORALEE CLEVELAND, *Secretary,*
City Board of Health,
Jacksonville, Fla.

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## THE NORTH DAKOTA BOARD OF DENTAL EXAMINERS

The North Dakota Board of Dental Examiners will hold its next meeting January 13-

16, at the Gardner Hotel, Fargo. All applications must be in the hands of the secretary by January 3.

GILBERT MOSKAU, *Secretary,*
Grand Forks.

## PENNSYLVANIA STATE BOARD OF DENTAL EXAMINERS

The Pennsylvania State Board of Dental Examiners will hold its next examination, December 10-13, at Philadelphia and Pittsburgh. Applicants should apply to the Department of Public Instruction, Harrisburg, for the necessary blanks, and on return of blanks with fee and photographs at least ten days before the date of examination, instructions will be sent to applicant and place of examination stated.

W. A. MCCREADY, *Secretary,*
1115 Highland Bldg.,
Pittsburgh.

## GREATER NEW YORK DECEMBER MEETING FOR BETTER DENTISTRY

The sixth Greater New York December Meeting for Better Dentistry will be held at the Hotel Pennsylvania, New York City, December 1-5. As in former years, there will be scientific papers, lecture or teaching clinics, topic discussions, scientific section meetings and general clinics. A special feature of this year's meeting will be one full afternoon devoted to the economic side of dentistry. At this session, efficient office conduct will be presented, also information on methods of saving and investments for professional men.

All members of the American Dental Association are eligible to register, registration fee, $5. A subscription blank and list of clinics will be ready for distribution about November 15.

There will be a manufacturers' exhibit in the hotel during the entire meeting.

## CHICAGO DENTAL SOCIETY MIDWINTER CLINIC

The sixty-seventh annual meeting of the Chicago Dental Society will be held at the Stevens Hotel, Chicago, February 2-5. Because of the great demand last year, the transactions of this meeting will be bound and made available at cost to those who wish them.

The exhibition hall of the Stevens Hotel has again been reserved by the Exhibit Committee for manufacturers' and dealers' exhibits.

The Society extends a cordial invitation to all members of the American Dental Association to attend.

HARRIS W. MCCLAIN, *President.*
HOWARD C. MILLER, *Secretary.*

## KINGS COUNTY MIDYEAR MEETING

The second midyear meeting for progressive dentistry, under the auspices of the Kings County Dental Society, will be held at the new St. George Hotel, in Brooklyn, February 25-28. Sessions have been arranged for operative clinics, table demonstrations, lectures, topic discussions, etc. Other features are: 1. Exhibit by manufacturers, dealers and laboratories. 2. Health demonstration by N. Y. Department of Health and N. Y. Tuberculosis Association. 3. Art exhibit. Admission is by registration, no fee required. A banquet will be given Saturday night, February 28.

JOSEPH NEMSER, *Chairman,*
Publicity Committee.

## KINGS COUNTY (N. Y.) DENTAL SOCIETY

The next meeting of the Kings County Dental Society will be held November 13, at the building of the Medical Society of the County of Kings, 1313 Bedford Ave., Brooklyn. Edwin N. Kent, of Boston, Mass., will lecture at 8:30 p. m. on "Dentistry as a Business." Clinics will precede the lecture.

J. L. FELSENFELD, *President,*
H. AUSUBEL, *Chairman,*
Educational Committee.

## RED CROSS ROLL CALL

The greatest Roll Call that has ever been conducted, to enroll 5,000,000 members in the American Red Cross in this country, will be opened November 11, Armistice Day, and will continue until Thanksgiving Day. In Chicago, the Red Cross will seek 225,000 members. Campaigns will be conducted in schools, offices, churches, theaters, banks and business by volunteer workers. The Red Cross needs the money these memberships will bring in to continue its tremendous work during 1931. The organization, which is a

semiofficial government service, is headed by the President of the United States of America. During the year ending June 30, the Red Cross nationally expended $1,182,350.08 for service and disabled exservice men and their families. In Chicago, $76,238 of this was distributed. The need for veteran relief is increasing and the climax is still thought to be many years away. The national organization handled 13,217 claims. Furthermore, $1,341,620.65 was needed to take care of 95,000 sufferers in 108 major disasters during the past year. More than $650,000 of this was in excess of contributions received for disaster relief. Fifty-one thousand and forty-seven Red Cross nurses were enrolled to meet national and international emergencies; 74,437 people were trained and certified in first aid work; 52,798 were certified in life saving; 37,626 were trained and certified in home hygiene and care of the sick. Nearly 7,000,000 school children were enrolled in the Junior Red Cross during the past year.

## CONNECTICUT STATE DENTAL ASSOCIATION

The Fourth Midseason Clinic of the Connecticut State Dental Association will be held Wednesday, November 5, at the Medical Association Bldg., 362 Whitney Ave., New Haven.

A. E. CARY, *President.*
C. E. PETERSON, *Sec'y-Treas.*

## TEXAS STATE BOARD OF DENTAL EXAMINERS

The next meeting of the Texas State Board of Dental Examiners will be held in Dallas, December 3. Applications must be in the hands of the secretary two weeks before the date of the examination.

MAXWELL C. MURPHY, *Sec'y-Treas.*
Temple.

## ODONTOLOGICAL SOCIETY OF WESTERN PENNSYLVANIA

The Odontological Society of Western Pennsylvania will meet at the William Penn Hotel, Pittsburgh, November 20-22. The essayists and their subjects are as follows: C. L. Drain, Iowa City, Iowa: "Dietary Control of Dental Caries"; George B. Winter, St. Louis, Mo.: "Extraction"; S. Harold Supplee, New York City: "Denture Materials"; Mr. Krogg, Rochester, N. Y.: "Radiography." John G. Logan, of Portage, Pa., will present special clinics. Educational sessions have been arranged for operative clinics, table demonstrations and topic discussions. A manufacturers' exhibit will be held in the hotel during the meeting. Entertainment for the families of attending members will be arranged. A cordial invitation is extended to all members of the American Dental Association.

W. HARRY ARCHER, *Chairman,*
Publicity Committee,
4052 Jenkins Arcade,
Pittsburgh.

## SOME RECENT BOOKS*

Available for Circulation from the

LIBRARY BUREAU

of the

AMERICAN DENTAL ASSOCIATION

58 E. Washington St., Chicago, Ill.

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|---|---|
| American Pharmaceutical Assn. | Pharmaceutical recipe book. Baltimore, Md., American Pharmaceutical Assn., 1929. |
| Anderson, G. M. | Orthodontic laboratory manual. St. Louis, Mosby, 1930. |
| Anthony, L. P. | Dental terminology. Chicago, American Dental Assn., 1930. |
| Appleton, J. L. T., Jr. | Bacterial infection with special reference to dental practice. Philadelphia, Lea & Febiger, 1925. |
| Appleton, J. L. T., Jr., & Bryant, C. K. | Laboratory guide in bacteriology. Philadelphia, Lea & Febiger, 1928. |
| Barail, L. C. | High-frequency in dentistry. Paris, France, La Verrerie Scientifique, 1927. (Pamphlet.) |
| Bartels, H. A. | Outline of bacteriology. New York, W. A. Broder, 1929. |
| Berger, Adolph | Principles and technique of oral surgery. Brooklyn, Dental Items of Interest Pub. Co., 1927. |
| Berger, Adolph | Principles and technique of the removal of teeth. Brooklyn, Dental Items of Interest Pub. Co., 1929. |
| Bilpodiwala, M. C. | Care of the teeth, or layman's handbook of dentistry. Bombay, India, Author, 1927. |
| Blunt, Katharine, & Cowan, Ruth | Ultraviolet light and vitamin D in nutrition. Chicago, University of Chicago Press, 1930. |
| Bödecker, C. F. | Elementary histology for dental hygienists. New York, W. A. Broder, 1929. |
| Box, H. K. | Necrotic gingivitis. Toronto, University of Toronto Press, 1930. (Pamphlet.) |
| Box, H. K. | Treatment of the periodontal pocket. Toronto, University of Toronto Press, 1928. |
| Bregstein, S. J. | Business conduct of an ethical practice. Brooklyn, Dental Items of Interest Pub. Co., 1929. |
| Broderick, F. W. | Dental medicine. St. Louis, Mosby, 1928. |
| Broderick, R. A. | Dental bacteriology. Outlines of dental science. Vol. 2. Edinburgh, Livingstone, 1926. |
| Brothers, E. D. | Dental jurisprudence. St. Louis, Mosby, 1928. |
| Brown, A. J. | Dental metallurgy. Outlines of dental science. Vol. 11. New York, Wood, 1928. |
| Brown, W. L. | Endocrines in general medicine. St. Louis, Mosby, 1927. |
| Buckley, J. P. | Modern dental materia medica, pharmacology and therapeutics. Philadelphia, Blakiston, 1926. |

*Books may be borrowed by members of the Association for a period of one week after date of receipt; and if not reserved for some one else, the period will be extended on request. A deposit of $2.00 for each volume to be sent at one time should accompany all requests for books. This will be refunded on return of books.

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| Bulleid, Arthur | Textbook of bacteriology for dental students. St. Louis, Mosby, 1927. |
| Bunting, R. W. | A text-book of oral pathology for students and practitioners of dentistry. Philadelphia, Lea & Febiger, 1929. |
| Burchard, H. H., & Inglis, O. E. | Textbook of dental pathology and therapeutics for students and practitioners. Philadelphia, Lea & Febiger, 1926. |
| Burkhart, H. J. | Care of the mouth and teeth. (National Health Series.) New York, Funk & Wagnalls, 1928. |
| Buxton, J. L. D. | Dental pathology. Outlines of dental science. Vol. 7. New York, Wood, 1927. |
| Buxton, J. L. D. | Dental surgery. Outlines of dental science. Vol. 8. New York, Wood, 1927. |
| Cahn, L. R. | Basic principles of general and oral pathology. Brooklyn, N. Y., Dental Items of Interest Pub. Co., 1929. |
| Campbell, J. M. | Those teeth of yours. London, Heinemann, 1929. |
| Cattell, Psyche | Dentition as a measure of maturity. Cambridge, Harvard University Press, 1928. |
| | Chapter of child health. . . . Commonwealth Fund child health demonstration in Clarke County and Athens, Georgia, 1924-1928. New York, Commonwealth Fund, Division of Publications, 1930. |
| Clapp, G. W. | Life and work of James Leon Williams. New York, Dental Digest, 1925. |
| Clark, C. A. | Dental radiography. Outlines of dental science. Vol. 5. New York, Wood, 1926. |
| Colyer, Stanley | Chronic infection of the jaws: a short radiological and clinical study. New York, Macmillan, 1926. |
| Daukes, S. H. | Medical museum: Modern developments, organization and technical methods based on a new system of visual teaching. London, Wellcome Foundation, Ltd., n.d. |
| Davis, M. M. | Clinics, hospitals and health centers. New York, Harper, 1927. |
| Dental Board of the United Kingdom | Four lectures on "general anaesthesia." London, Dental Board of the United Kingdom, 1926. |
| Dental Board of the United Kingdom | Three lectures on "dental alloys." Delivered under the auspices of the Dental Board of the United Kingdom. London, Dental Board of the United Kingdom, 1927. |
| Diamond, M. | Dental anatomy. A graphic presentation of tooth forms with an original technique for their reproduction. New York, Macmillan, 1929. |
| Endleman, Julio | Special dental pathology: a treatise for students and practitioners. St. Louis, Mosby, 1927. |
| Federspiel, M. N. | Harelip and cleft palate. St. Louis, Mosby, 1927. |
| Feldman, M. H. | Manual of exodontia. Philadelphia, Lea & Febiger, 1930. |
| | First International Orthodontic Congress: held at New York City, August 16-20, 1926. St. Louis, Mosby, 1927. |
| Fones, A. C. | Mouth hygiene; a textbook for dental hygienists. Philadelphia, Lea & Febiger, 1927. |
| Fones, A. C. | Preventive dentistry. Philadelphia, Lea & Febiger, 1925. |
| Foote, J. S. | Bone as a measure of development: When and how we acquired our teeth. Chicago, American Dental Association, 1928. |
| Franzen, Raymond | Public health aspects of dental decay in children. New York, American Child Health Assn., 1930. |
| Gies, W. J. | Dental education in the United States and Canada. New York, Carnegie Foundation, 1926. |
| Goepp, R. M. | Dental state board questions and answers. Philadelphia, Saunders, 1928. |

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| Goslee, H. J. | Principles and practice of crown and bridgework. Brooklyn, Dental Items of Interest Pub. Co., 1926. |
| Greenfield, A. L. | Interpretation of dental radiograms. Rochester, N. Y., Ritter Dental Mfg. Co., 1926. |
| Gregory, W. K. | Our face from fish to man. New York, Putnam, 1929. |
| Guerini, Vincenzo | Life and works of Guiseppangelo Fonzi. Philadelphia, Lea & Febiger, 1925. |
| Gwathmey, J. T. | Textbook of anesthesia. New York, Macmillan, 1929. |
| Gwinn, C. D. | Textbook of exodontia. Philadelphia, Lea & Febiger, 1927. |
| Gysi, Alfred | Special teeth for cross-bite cases. New York, Dentists' Supply Co., 1928. (Pamphlet.) |
| Hackh, I. W. D. | Chemical reactions and their equations. Philadelphia, Blakiston, 1928. |
| Haden, R. L. | Dental infection and systemic disease. Philadelphia, Lea & Febiger, 1928. |
| Hanau, R. L. | Dental engineering. Buffalo, N. Y., Author, 1926. |
| Hanau, R. L. | Hanau intraoral method vs. Gysi extraoral method. Buffalo, N. Y., Author, 1927. |
| Hartman, L. L. | Teeth and the mouth. New York, Appleton, 1927. |
| Head, Joseph | Everyday mouth hygiene. Philadelphia, Saunders, 1925. |
| Headridge, David, & Gibson, S. K. | Dental anatomy, human and comparative. Outlines of dental science. Vol. 6. New York, Wood, 1928. |
| Hedstrom, Gustav | Gangrene of dental pulp and its treatment. Stockholm, Sweden, Isaac Marcus, 1927. |
| Herbst, Emil, & Apffelstaedt, Max | Atlas and grundriss der missbildungen der kiefer and zähne. München, J. F. Lehmanns, 1928. |
| Hertzler, E. | Technic of local anesthesia. St. Louis, Mosby, 1925. |
| Hess, Walter, & Zurcher, Ernst | Anatomy of the root-canals of the teeth. New York, Wood, 1925. |
| Hogeboom, F. E. | Practical pedodontia or juvenile operative dentistry and public health dentistry. St. Louis, Mosby, 1927. |
| Hyatt, T. P. | Hygiene of mouth and teeth. Brooklyn, N. Y., Brooklyn Dental Pub. Co., 1929. |
| Jamieson, J. D. H. | Operative dentistry. Outlines of dental science. Vol. 9. New York, Wood, 1927. |
| Jerman, E. C. | Modern x-ray technic. St. Paul, Bruce Pub. Co., 1928. |
| Jordan, M. Evangeline | Operative dentistry for children. Brooklyn, Dental Items of Interest Pub. Co., 1927. |
| Kanner, Leo | Folklore of the teeth. New York, Macmillan, 1928. |
| Kells, C. E. | Dentists own book, including a complete bookkeeping and recording system. St. Louis, Mosby, 1925. |
| Kells, C. E. | Three score years and nine. New Orleans, The Author, 1926. |
| Kennedy, Edward | Partial denture construction. Brooklyn, N. Y., Dental Items of Interest Pub. Co., 1928. |
| Kent, E. N. | Business side of dentistry. St. Louis, Mosby, 1929. |
| LeGro, A. L. | Ceramics in dentistry. Brooklyn, Dental Items of Interest Pub. Co., 1926. |
| Leriche, R., & Policard, A. | Normal and pathological physiology of bone. St. Louis, Mosby, 1928. |
| Livingston, Alexander | Dental histology. Outlines of dental science. Vol. 10. New York, Wood, 1928. |
| Logan, J. D. | Dental prosthetics. Outline of dental science. Vol. 4. Edinburgh, Livingstone, 1926. |
| Marriott, W. M. | Infant nutrition: a textbook of infant feeding for students and practitioners of medicine. St. Louis, Mosby, 1930. |
| Marsden, P. H. | Dental materia medica. Outlines of dental science. Vol. 3. Edinburgh, Livingstone, 1926. |

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| Maximow, A. A., & Bloom, Wm. | Textbook of histology. Philadelphia, Saunders, 1930. |
| McCollum, E. V., & Simmonds, Nina | Food, nutrition and health. Baltimore, Authors, 1928. |
| McCollum, E. V., & Simmonds, Nina | Newer knowledge of nutrition. New York, Macmillan, 1929. |
| McCoy, J. D. | Applied orthodontia. Philadelphia, Lea & Febiger, 1927. |
| McGaw, Emma J. | Dental assistant. St. Louis, Mosby, 1926. |
| McGehee, W. H. O. | Textbook of operative dentistry. Philadelphia, Blakiston, 1930. |
| Marshall, J. A. | Diseases of the teeth; their diagnosis and treatment. Philadelphia, Lea & Febiger, 1926. |
| Mead, S. V. | Diseases of the mouth. St. Louis, Mosby, 1928. |
| Mellanby, May | Diet and the teeth: an experimental study. Part I. Dental structure in dogs. London, His Majesty's Stationery Office, 1929. |
| Merritt, A. H. | Periodontal diseases: diagnosis and treatment. New York, Macmillan, 1930. |
| Moorehead, F. B., & Dewey, Kaethe W. | Pathology of the mouth. Philadelphia, Saunders, 1925. |
| Mustard, H. S. | Cross-sections of rural health progress. . . . Commonwealth Fund child health demonstration in Rutherford County, Tennessee, 1924-1928. New York, Commonwealth Fund, Division of Publications, 1930. |
| Nevin, Mendell, & Puterbaugh, P. G. | Conduction, infiltration and general anesthesia in dentistry. Brooklyn, Dental Items of Interest Pub. Co., 1927. |
| | New and nonofficial remedies, 1929. Chicago, American Medical Association, 1929. |
| | New and nonofficial remedies, 1930. Chicago, American Medical Association, 1930. |
| Nichols, I. G. | Prosthetic dentistry: an encyclopedia of full and partial denture prosthesis. St. Louis, Mosby, 1930. |
| Orban, Balint | Dental histology and embryology. Philadelphia, Blakiston, 1929. |
| Osgood, H. A. | Teeth and jaws roentgenologically considered. Annals of roentgenology, volume 5. New York, Hoeber, 1929. |
| Ottolengui, Rodrique | Table talks on dentistry. Brooklyn, Dental Items Interest Pub. Co., 1928. |
| Owre, Alfred | Prunes or pancakes. Minneapolis, University of Minnesota Press, 1926. |
| Perry, Maude A. | Dietetics and nutrition. St. Louis, Mosby, 1930. |
| Pollia, J. A. | Fundamental principles of aveolo-dental radiology. Brooklyn, Dental Items of Interest Pub. Co., 1930. |
| Posner, J. J. | Minor oral surgery simplified. Philadelphia, Patterson & White, 1928. |
| Prinz, Hermann | Dental formulary. Philadelphia, Lea & Febiger, 1930. |
| Prinz, Hermann | Dental materia medica and therapeutics. St. Louis, Mosby, 1926. |
| Prinz, Hermann | Diseases of soft structures of teeth. Philadelphia, Lea & Febiger, 1928. |
| Raper, H. R. | Clinical preventive dentistry. Rochester, N. Y., Ritter Dental Mfg. Co., 1926. |
| Rapp, F. W. | Elements of anatomical articulation or the mathematics of dental prosthetics. London, Bale, 1930. |
| Reidy, M. J. | This tooth proposition. Des Moines, Iowa, Author, 1927. |
| | Ritter practice building suggestions. Rochester, N. Y., Ritter Dental Mfg. Co., 1926. |
| Roberts, Lydia J. | Nutrition work with children. Chicago, University of Chicago Press, 1927. |
| Rose, Mary S. | Foundations of nutrition. New York, Macmillan, 1927. |

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| Schroff, Joseph | Fundamentals of pathology. New York, W. A. Broder, 1929. |
| Sears, V. H. | Full denture procedure. New York, Macmillan, 1929. |
| Shaw, D. M. | Dental prosthetic mechanics. London, Arnold, 1927. |
| Sicher, Harry, & Tandler, Julius | Anatomie für Zahnärzte. Vienna, Julius Springer, 1928. |
| Silverman, S. L. | Principles and practice of oral surgery. Philadelphia, Blakiston, 1926. |
| Simon, P. W. | Fundamental principles of a systematic diagnosis of dental anomalies. Boston, Stratford, 1926. |
| Simon, W., & Base, Daniel | Manual of chemistry. Philadelphia, Lea & Febiger, 1927. |
| Simpson, C. O. | Technic of oral radiography. St. Louis, Mosby, 1928. |
| Smith, G. F. R. | Dental anesthesia. Outlines of dental science. Vol. 1. Edinburgh, Livingstone, 1926. |
| Sorrin, Sidney, & Miller, S. C. | Practice of periodontia. New York, Macmillan, 1928. |
| Steadman, F. St. J. | Pyorrhea alveolaris. St. Louis, Mosby, 1927. |
| Stieglitz, Julius | Chemistry in medicine. New York, Chemical Foundation, 1928. |
| Stillson, W. C. | Dental anatomy: classroom and laboratory quiz compend with supplementary notes. Philadelphia, Saunders, 1929. |
| Stoloff, C. I. | Your teeth. New York, Dutton, 1929. |
| Suter, A. B. W. | Dentistry—a profession and a business. Rochester, N. Y., Ritter Dental Mfg. Co., 1930. |
| Swendiman, G. A. | Dental health. Boston, Stratford Co., 1930. |
| Tanguy, T. V. | Science and practice of actinotherapy. St. Louis, Mosby, 1928. |
| Turner, C. R., & Anthony, L. P. | American textbook of prosthetic dentistry. Philadelphia, Lea & Febiger, 1928. |
| Turner, Clair E. | Personal and community health. St. Louis, Mosby, 1930. |
| Tyree, C. | Dental health in the prevention of disease. London, Kentucky, Author, 1927. |
| Wallace, J. S. | Physiology of oral hygiene and recent research. London, Bailliere, Tindall & Cox, 1929. |
| Wallace, J. S. | Teeth and health. London, Faber & Gwyer, 1926. |
| Wallace, J. S. | Variations in the forms of the jaws, with special reference to their etiology and their relation to the occlusion of the dental arches. New York, Wood, 1926. |
| Wang, C. Y. | Handbook of pathology. New York, Wood, 1925. |
| Ward, L. (Ed.) | American textbook of operative dentistry. Philadelphia, Lea & Febiger, 1926. |
| Weinberger, B. W. | Orthodontics: an historical review of its origin and evolution. 2 vols. St. Louis, Mosby, 1926. |
| Wells, F. M. | Sound teeth in a sound body. Montreal, Author, 1926. |
| Winter, G. B. | Principles of exodontia, as applied to the impacted mandibular third molar. St. Louis, American Medical Book Co., 1926. |
| Winter, Leo | Textbook of exodontia. St. Louis, Mosby, 1927. |
| Woodbury, C. E. | Making and filling of cavities in the proximal surface of the front teeth with gold foil. Omaha, Nebr., Author, 1929. |
| Zemsky, J. L. | Oral diseases: practical treatise offering diagnostic and therapeutic aid to the practitioner of medicine and dentistry. Brooklyn, N. Y., Physicians and Surgeons Book Co., 1930. |
| Zoethout, W. D. | Textbook of physiology. St. Louis, Mosby, 1928. |

A list of the package libraries was published in the October, 1930, issue of THE JOURNAL.

# OFFICERS AND TRUSTEES OF THE AMERICAN DENTAL ASSOCIATION

## 1930-1931

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Ernest W. Elmen.....608 Sioux Falls Nat'l Bank Bldg., Sioux Falls, S. D.

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Harry B. Pinney.....58 E. Washington St., Chicago, Ill.

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